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Volume XVII Toronto, February, 1940 Number 4

The University of Toronto Medical Journal

(AN UNDERGRADUATE PUBLICATION)

This journal is published six times a year, November to April, by the Medical Society of the University of Toronto, to serve as a medium for student expression and to bring to the student recent articles of scientific interest.

$1.50 per volume. 25c. per single copy.

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Editorial

TYPHOID MARY

Mary Mallon was the most famous typhoid carrier the world has known. Her discovery brought to light for the first time a chronic typhoid carrier with infected faeces. This type of carrier is now regarded as by far the most common. The credit for her discovery goes to Dr. George A. Soper who has published the story of her curious career in the *Bulletin of the New York Academy of Medicine*, upon which this short article is based.

In 1906 a New York banker rented a summer home at Oyster Bay, New York. In August of that year, eleven persons in the household were taken suddenly ill with typhoid fever. The well, the cesspool, the food supplies and the bathing water were all investigated before Dr. Soper turned his attention to carriers and by a process of exclusion came to the cook who had left soon after the outbreak. He went to the employment agency which had placed the cook and obtained a list of the families with which Mary had worked. A study of these revealed a

series of seven household epidemics. The cook in no case was suspected.

One year after the Oyster Bay epidemic, Mary was working as cook in an old-fashioned house on Park Avenue. The laundress and an only child were seriously ill with typhoid. It was here that Dr. Soper finally caught up with typhoid Mary, but the interview was an unsatisfactory one because his request for samples of urine, blood and faeces were repelled with a carving fork. She denied ever having had typhoid fever.

A recommendation was made to the New York City Health Department that Mary Mallon be taken into custody. An inspector was sent to collect the necessary specimens, but the interview was again cut short by a hastily slammed door.

Next morning a Department of Health ambulance drew up quietly, with an inspector and three policemen. Two policemen were stationed to prevent her escape and the third accompanied the inspector to the door. Mary answered the knock, but upon learning the identity of her visitors she ran back to the kitchen and disappeared. A three hour search finally revealed her in an outside closet with ashcans piled high against the door. She struggled and cursed until finally the policemen picked her up and put her in the ambulance. Mary was locked in one of the isolation wards of a New York hospital and a few days later the bacteriological report showed that her faecal material contained a pure culture of *B. typhosus*. She was given a small bungalow on North Border Island near the Riverside Hospital. Her food was brought to her, which she cooked and ate alone.

Two years later Mary sued the city for release under Habeas Corpus proceedings, but the judge dismissed the case because Mary had refused the surgical removal of her gall bladder and the Court was unwilling to take the responsibility of releasing her. However, a year later she was released on her pledge to give up her vocation as a cook and to report to the Department of Health every three months. On her release she disappeared, changed her name and went back to cooking for restaurants, hotels and sanatoria.

One day, five years later, Dr. Soper received a 'phone call from a staff doctor of the Sloane Hospital for Women to the effect that he had a typhoid epidemic of more than twenty cases on his hands. The other servants had jokingly nicknamed the cook "Typhoid Mary" and Mary Mallon it was. She was sent back to her bungalow where she remained for twenty-three years. During this period she never once tried to escape. She was given a job in the laboratory of the hospital and learned to make simple routine tests.

On Christmas morning, 1932, Mary was found paralyzed on the floor of her bungalow by a delivery boy. She had suffered a stroke which left her powerless to walk, and for the remaining six years of her life she was taken care of in hospital.

Mary Mallon died on November 11th, 1938. No autopsy was performed, but her death certificate stated death was due to terminal broncho-pneumonia following chronic nephritis and chronic myocarditis, and that the deceased had been a typhoid carrier for twenty-four years. At the funeral service at St. Luke's in the Bronx there were only nine persons present, and there was not one who followed her to the grave.

The total number of typhoid cases traced to Mary Mallon was fifty-three, with three deaths. This is not a large toll compared to the records of some carriers who have since been found, but there doubtless were many cases produced by Mary that never came to light.

PYURIA

By J. C. McCLELLAND, B.A., M.B., F.R.C.S. (C.)
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What would you do for the first patient to enter your office who was found to have pus cells in the urine? This problem has been faced by many a physician and he must have a good general idea of the common causes of pyuria and the proper methods of investigation to give the patient an intelligent opinion as to the best procedure to reach a diagnosis.

Collection of Urine

The urine is obtained by having the male patient pass it into two sedimentation glasses. The small quantity in the first glass contain the washing from the urethra and the second glass contains the urine from the bladder and is comparatively free from the abnormal elements found in the urethra. These two glasses are examined. If pus is found in the first glass, its origin is said to be from the urethra or prostate, while pus in the second glass may come from the bladder or the kidneys. Urine from a female patient should be collected by catheter if pus cells are found in the ordinary voided specimen.

Examination of Urine

Each sample of urine should have the routine chemical tests applied, followed by a microscopic examination of the sediment found after centrifuging. Pus cells are recognized as circular bodies filled with granules. If acetic acid has been added, these granules dissolve, leaving the nucleus standing out prominently. These cells are slightly larger than red blood cells.

It occasionally happens that the specimen is cloudy to the eye and one recognizes that the cloud is due either to salts or the cellular elements. By adding acetic acid the cloud caused by phosphates clears, but if it remains cloudy the specimen may be heated. If urates are causing the cloud they will disappear with heat. Thus the routine for examining a cloudy specimen of urine should be to acidify with acetic acid and if the cloud does not disappear, it should be heated. Following this, the sediment is examined under the microscope.

Physical Examination

A routine physical examination should be done of all systems. The urinary tract is examined by abdominal palpation, including the flanks, kidney areas, external genitalia and areas of tenderness noted, also the presence of any abnormal swelling. Rectal examination should include the area around the sphincter and the prostate. When the rectum and prostate have been palpated, the prostate can be pressed gently and some secretion expressed into the urethra for microscopic examination of the wet film for the presence of pus cells. Frequently, this is found to be the source of the pus.

X-ray

It happens commonly that some symptom such as pain suggests the kidney as the site of the lesion and the origin of pus. If so, a plain roentgenogram of the kidneys, ureters and bladder is of help. It will show the presence or absence of a stone and frequently some variation in the size of the kidneys. An intravenous urogram may show some abnormalities in the urinary tract but it is the ureteral catheter which is essential to show the origin of pus cells.

Cystoscopy

This is a special examination and should not be done until as much of the above information is obtained as possible.

Bladder inspection is carried out with a study of the bladder neck, the

trigone, ureteral orifices and bladder mucosa. The presence of enlargement of the prostate gland, vesical calculi, new growths, localized areas of inflammation, tubercles, foreign bodies and the orifices of diverticula are seen. Residual urine is measured also.

Ureteral catheters are passed up both ureters by means of which the individual kidney urines can be collected and examined for the presence of pus cells and organisms. The relative functional efficiency of the kidneys can be studied by phenolphthalein or indigocarmine tests. A retrograde pyelogram can be done by the injection of 12% sodium iodide to the kidney pelves. This will give an X-ray outline of the upper urinary tract. In this way, any variation from the normal size or shape of the pelvis can be noted. A shadow in the kidney area can be localized as inside, in the case of calculi, or outside, as seen frequently with calcified glands simulating renal stones.

Differential Diagnosis

Having had specimens of urine collected separately from the bladder and each kidney at cystoscopy, the origin of the pus can be stated at once. Both kidney specimens may be entirely free from pus and the bladder specimen contains some. Again, one kidney specimen or both kidney specimens may contain pus cells. One must not overlook the prostate as the possible source of the pus by bearing in mind the result of the examination of the prostatic secretion at the time of the rectal examination. Once the source of the pus is determined accurately by the methods outlined, one can proceed with a considération of the lesions in the kidney, ureter, bladder, prostate or urethra, which are accustomed to show pus cells in the urine.

The physician must bear in mind the group of lesions which may occur in any organ of the body as represented by the following table.

A. Congenital.

B. Acquired.
   (1) Inflammations—acute or chronic.
   (2) Tuberculosis.
   (3) Syphilis.
   (4) New Growth.
   (5) Trauma.
   (6) Calculus.
   (7) Cysts.
   (8) Actinomycosis.
   (9) Blastogenesis.

These lesions must be applied to each organ making up the urinary tract in the following way.

Kidney

A. *Congenital.* Certain congenital abnormalities manifest themselves in the first few years of life and result in the finding of pus cells in the urine. Dilatation of the bladder may be found secondary to congenital valves of the posterior urethra. Hydroureters, together with a hydronephrotic dilatation of the kidneys are also seen. Renal calculi are found in the first few years of life and it has been suspected that they were present before birth. The earliest I have seen was in a boy two years of age who had multiple stones in both kidneys. Polycystic disease of the kidneys is recognized mostly in adults as it does not give symptoms during early life. The so-called "pyelitis" of a child may come from most of the conditions present in the adult and the child should have a complete urinary tract investigation if the pus does not disappear readily with antiseptics or shows a tendency to recur repeatedly.

B. *Acquired.*
(1) *Inflammation*
   (a) Pyelonephritis—acute or chronic.
   (b) Pyonephrosis.

These conditions are found secondary to some other infection; to a calculus, a new growth or an obstruction. The diagnosis is made by obtaining urine direct from the kidney with a catheter. Pyonephrosis is a massive destruction of the kidney parenchyma, producing a large quantity of pus from a large kidney which can be palpated usually in the flank.

(2) *Tuberculosis.* The young adult who has an increasing frequency of

urination should be suspected of tuberculosis. Usually, frequency is the only symptom. If pus cells are found in the urine and in a smear no organisms are found, then one must suspect tuberculosis and do an acid fast stain of the centrifuged sediment. With this test, the bacteriologist should find the organisms in approximately 70%. In the remaining ones the tubercle bacillus is detected by cultures and guinea pig inoculations. Again, the ureteral catheter must be used to find the origin of the pus. The pyelogram is a great help in showing up the cavities produced in tuberculous kidneys. An intravenous pyelogram may show no excretion from an advanced lesion in the kidney or may show the cavitation.

(3) *Syphilis* of the kidney is rare and does not produce pus.

(4) *New Growth.* New growths usually have haematuria as their symptom. Occasionally pus cells are found but it is unusual.

(5) *Trauma.* There is a recent history of injury to the kidney area with the passage of blood. In rare instances the kidney becomes infected producing a large quantity of pus.

(6) *Calculus.* The usual symptom is pain in the flank and radiating down the course of the ureter. Some larger stones do not give any symptom but pus is found in the urine coming from the kidney containing the stone. A calculus is the contributing agent to most of the pyonephroses.

(7) *Cysts.* Cysts in the kidney do not produce pus.

Nephroptosis. This condition usually has a mild pyelonephritis associated with it which may become acute. Its diagnosis is made by noting the excursion of the kidney in the erect position as shown by the erect pyelogram. The moveable kidney can be felt in the flank.

Hydronephrosis. A hydronephrosis does not usually have pus cells coming from it. A few instances of a few pus cells are found and these become pyohydronephroses, in which there is an associated pyelonephritis.

Ureter

Calculus. A stone in the ureter is the main cause of trouble here. There may be no pus present in the early stages but later it will come and if the ureter becomes obstructed it will produce a pyelonephritis. It is associated with pain along the course of the ureter and is diagnosed by cystoscopy and the X-ray.

Stricture. A narrowing along any portion of the ureter may occur and give the same symptoms as the calculus. Sooner or later infection comes and shows itself by pus from that ureter.

Bladder

Inflammations. Acute or chronic cystitis may be present and shown by frequency, dysuria, haematuria or any one of the urinary symptoms. It is usually secondary to some obstruction at the neck of the bladder, a foreign body such as a stone, or some injury.

Tuberculosis. Tuberculosis of the bladder is secondary to a renal lesion or to a genital tuberculosis. Further, a genital tuberculosis is usually a manifestation of a renal tuberculosis, though we have patients with a tuberculous epididymitis and a cystitis without repeated examinations of the kidneys showing tuberculosis in the kidney. Pus cells and tubercle bacilli are present and can be demonstrated in practically all cases.

New Growths. Neoplasms in the bladder have haematuria as the first symptom, but it is occasionally frequency. Pus cells and pyogenic organisms may be present in large quantities. In the terminal stages of bladder tumours there is an advanced pyelonephritis or pyonephrosis.

Calculus. Stones in the bladder are fairly common. Infection comes in and the cystitis produces pus cells in the urine.

Cysts. Cysts at the bladder neck produce obstruction and a cystitis. Also, there may be numerous small cysts over the surface of the bladder as an evidence of an old standing cystitis, called cystitis cystica.

Diverticulum. Diverticula are sacs or herniae of the bladder mucosa through

the bladder wall. They may be small or as large as the bladder itself. They are associated with a large quantity of pus in the urine.

Prostate

Inflammation. An acute inflammation of the prostate may give a good deal of cystitis, due to its obstruction which may be complete, causing retention. Chronic inflammation of the prostate gives a few shreds in the urine and a small quantity of pus. The diagnosis is made by massage of the gland and finding pus cells in the prostatic secretion. The urine will contain pus in varying amounts.

Tuberculosis. Tuberculosis of the prostate is usually symptomless by itself. It is sometimes recognized on rectal examination alone, but is most often found when a tuberculous epididymitis is present.

New Growth. Enlargement of the prostate, whether benign or malignant, produces an obstruction to the free outflow of urine from the bladder. This in turn causes a cystitis and the production of pus in the urine.

Calculus. Stones in the prostate are usually in large numbers, the size of small buck-shot. Infection in the prostate is present and they present the picture of a chronic prostatitis.

Urethra

Inflammations. Acute inflammation in the urethra is shown by the production of pus coming from the urethra. This may be due to the gonococcus or non-specific organisms. Some of the pus should be examined in a smear to determine the exact cause.

Chronic inflammation in the urethra shows itself by shreds and pus cells in the first glass of urine voided. A stricture of the urethra is a common cause. The prostate usually has an associated chronic inflammation and should be examined.

Calculus. Stones may be stuck in the urethra and produce inflammation due to their obstruction and injury and thus produce pus cells.

Conditions Outside the Urinary Tract Producing Inflammation in the Tract

Any pelvic or appendiceal inflammation or abscess formation which lies against the bladder may give a cystitis, or in rare instances, the abscess may rupture into the bladder. New growths of the rectum or cervix invade the bladder and produce a cystitis and pus in the urine.

Treatment

The treatment for these conditions is one of removing the cause first and the infection should clear. If no cause for the infection is found, the usual urinary antiseptics now in use should be tried.

Conclusions

1. In all cases of pus cells in the urine, investigate the cause.
2. Each specimen of urine showing pus cells should have a smear made and examined for organisms. If none are found the urine should be centrifuged at high speed and the sediment examined for tubercle bacilli. Cultures for tubercle bacilli and guinea pig inoculations should be done also if indicated.
3. If a stone is suspected, an X-ray of the urinary tract should be taken.
4. Treatment will depend on a correct diagnosis of the origin of the pus cells.

THE EXPERIMENTAL PRODUCTION OF ARTERIAL HYPERTENSION

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Richard Bright in 1827 was the first to recognize that high blood pressure was of frequent occurrence in the disease which now bears his name. There were, of course, no clinical means of measuring blood pressure in Bright's day but, from the hypertrophy of the left ventricle so frequently observed in chronic nephritis, he shrewdly deduced that high arterial blood pressure existed. The association of kidney disease with hypertension soon became firmly established in clinical teaching. Allbutt pointed out many years later that hypertension also occurs in the absence of kidney disease and apparently independently of any other diseased state. He also insisted that arteriosclerosis was not a cause of, but was not infrequently caused by arterial hypertension. Allbutt gave the term *hyperpesia* to the high blood pressure which occurs in persons with apparently healthy kidneys. This form of the disease is now more usually referred to as *essential* or *primary hypertension*. Whether occurring in association with kidney disease or in persons otherwise in good health, the cause of the hypertension in terms of the physiology of the vascular system is an increase in the peripheral resistance—that is, an inordinate degree of constriction of the systemic arterioles. The blood pressure in the pulmonary circuit and in the capillaries and veins of the systemic system is not elevated. The manner in which the vasoconstriction is brought about has been a topic of much speculation, but the work of Goldblatt and his associates has provided in the writer's opinion evidence of a most convincing character that interference with the blood supply to the kidney is the primary factor in the production of the hypertension in chronic Bright's disease. Goldblatt induced ischaemia of the kidney in dogs by means of a specially devised adjustable silver clamp which partially occluded the renal arteries. When a clamp was applied to the renal artery of one side the arterial pressure rose within two or three days by about 50 mm. Hg (systolic) and remained elevated for three or four weeks, but then returned to normal. If the arteries of both kidneys were constricted or if one kidney was rendered ischaemic and its fellow removed, the blood pressure rose to a higher level and showed no tendency to return to normal. In animals so treated over five years ago, hypertension persists.

If the constriction of the renal arteries is moderate the kidneys show little change from the normal and there is no impairment of renal function. On the other hand, a severe degree of renal ischaemia is followed by pathological changes in the renal parenchyma, consisting chiefly of tubular degeneration or atrophy; renal insufficiency develops. In such animals, with renal insufficiency plus hypertension, severe degenerative and necrotic changes are found in the systemic arterioles closely comparable with those characteristic of malignant hypertension in man, except that the vessels within the kidney itself are not affected, since they are protected by the clamp on the renal artery from the high pressure which exerts such a destructive action upon the systemic arterioles.

Though Goldblatt was unable to produce permanent hypertension in dogs by means of renal ischaemia if the other kidney was left *in situ* with its blood supply intact, Wilson and Byrom in experiments on rats have succeeded in producing persistent hypertension, together with degenerative changes in the systemic vessels, by the constriction of one renal artery without interfering with the kidney of the opposite side. The vessels of the intact kidney shared in the degenerative changes.

The Mechanism Underlying the Production of Hypertension by Renal Ischaemia

There are two possible ways in which the hypertension associated with renal ischaemia might be produced, namely, (1) by means of a *vasomotor reflex* elicited through afferent nerve endings in the kidney, the vasomotor centre and the vasoconstrictor nerves, i.e., through a mechanism analogous to the carotid sinus reflex. (2) Through the liberation by the renal cells of a *chemical substance* —a humor or hormone—which might then act either (a) directly upon the systemic arterioles, (b) by bringing about the discharge of a pressor hormone, e.g., from the pituitary or the adrenal medulla, or (c) by an action, direct or indirect, upon the vasomotor centre.

It has been shown conclusively, however, that the mechanism is in no way a nervous one. Denervation of the kidney by the section of all nerve fibres entering it along the pedicle, cutting the splanchnic nerves or excision of the entire sympathetic chain, or even destruction of the spinal cord below the first thoracic segment, is without effect upon the response of the circulation to renal ischaemia, and in no way alters the course of the hypertensive state. Furthermore, if a kidney is excised and transplanted into the animal's neck (renal artery anastomosed to carotid, renal vein to jugular) and then rendered ischaemic, hypertension develops in the usual way (Blalock and Levy). The mechanism, then, is indubitably chemical in nature and quite independent of any nervous influence.

"Renin," the Pressor Substance Responsible for Renal Hypertension

In 1898 Tigerstedt and Bergman extracted the renal cortex of rabbits and obtained a product which upon intravenous injection exerted a powerfully hypertensive action. They gave the name "renin" to the active pressor principle, suggesting that in chronic nephritis it was produced in excess and entered the general circulation. Defective elimination of the substance in the urine and its accumulation in the blood is, of course, another possibility, but this seems to be refuted by an experiment performed by Geer and Dragstedt. They diverted the urine into the blood stream of dogs; hypertension did not result. That a pressor principle is present in the cortex of the kidney is a fact well established by recent work. The quantity of the material procurable from normal kidneys is nearly as great as that furnished by ischaemic renal tissue. This fact among others gives support to the belief that it is not a pathological product but in reality a physiological principle—a humor or hormone—elaborated by certain elements of the kidney and serving to increase the renal blood flow through raising the general blood pressure. The most likely renal elements possessing this endocrine function are those composing the "juxta glomerular apparatus" described recently by Goormaghtigh and his associates.

The passage of a hypertensive principle from the ischaemic kidney into the circulation has been demonstrated by Houssay and his colleagues. They introduced an ischaemic kidney removed from a hypertensive animal into the circulation of a nephrectomized but non-hypertensive animal. In 27 out of 30 animals upon which this grafting experiment was carried out, a rise in blood pressure was recorded. In a corresponding series of experiments in which the kidneys from apparently normal animals were grafted, no rise in blood pressure of the recipient animals occurred in the great majority of trials. Solandt, Nassim and Cowan have also detected the passage of a pressor principle from a hypertensive to a nephrectomized, non-hypertensive animal in experiments in which the blood of the former was transferred by a specially devised pump to the circulation of the latter. The hypertensive effect of a grafted ischaemic kidney or of blood transferred from a hypertensive animal can be demonstrated only upon one from which the kidneys have been excised. The explanation given for this fact is that normal renal tissue possesses the power to annul

the action of the pressor principle discharged by the ischaemic kidney. A highly purified preparation of renin has been obtained by Page and Helmer. This material is in the form of a crystalline picrate and exerts a powerfully hypertensive action upon injection into the circulation of intact animals, but is free from any vasoconstrictor activity when dissolved in saline and perfused through the isolated tail of a dog or ear of a rabbit. The saline solution becomes active, however, upon the addition of plasma or of red cells which, according to these workers, contain an activating substance (*renin activator*) of a protein nature. The active principle (i.e., renin plus renin activator) has been named *angiotonin*.

A Possible Relationship Between the Action of Renin and the Known Endocrine Organs

The pituitary, thyroid and adrenal medulla can be ruled out as playing any role in the development of renal hypertension. Neither hypophysectomy, thyroidectomy nor ablation of the adrenal medulla prevents the onset of hypertension following constriction of the renal arteries. On the contrary, the adrenal cortex appears to be implicated in some way, for Goldblatt found that adrenalectomized animals maintained in good health by means of a high sodium and low potassium diet did not give a hypertensive response to constriction of the renal arteries, but did so if treated with cortin. These observations indicate that renin exerts its effect directly upon the arterioles but that the hormone of the adrenal cortex is necessary to maintain the reactivity of the vascular muscle to its action.

The Importance of the Foregoing Experimental Results in Respect to the Cause of Clinical Hypertension

In chronic nephritis the walls of the afferent glomerular arterioles show degenerative changes which lead to partial or complete occlusion of many of these vessels. The vascular bed of the kidney is markedly contracted, the renal tissue no doubt being in a state of ischaemia closely comparable with that produced by experiment, except that in the latter it is the lumen of the main artery and not the calibres of the smaller vessels which is reduced. Few can doubt that renal ischaemia is the basis of the arterial hypertension in both instances. The severe degenerative changes described above as occurring in the systemic vessels are the result of the high and sustained hypertension and the counterpart of the systemic arterial damage found in malignant hypertension seen clinically. One naturally asks, "Has essential hypertension, that is, hypertension without renal insufficiency, its origin also in some abnormality of the renal circulation?" This is a much more difficult question, for in this form of the disease the histological appearance of the kidney is usually described as showing no definite departure from the normal. However, Moritz and Oldt in a recent investigation have secured evidence for the existence of renal arteriolar disease in essential hypertension. In a histological examination of the kidneys of 100 hypertensive cases in which there had been no clinical indication of renal disease, arteriolar damage, consisting of endothelial hyperplasia with hyaline degeneration and medial hypertrophy, was found without exception. In the same number of non-hypertensive cases, renal arteriolar disease was absent. So far as the systemic vessels were concerned, no corresponding distinction between hypertensive and non-hypertensive cases was observed. These findings indicate either that the walls of the renal vessels are peculiarly susceptible to damage by high pressure, which does not seem likely, or that the arteriolar changes, and a certain degree of renal ischaemia consequent thereto, constitute the primary cause of the hypertension.

The hypertension associated with renal ischaemia suggests, as already mentioned, a physiological mechanism which it is not difficult to conceive serves to proportion the renal blood flow to the work which the kidney is called upon to do. If such an idea is sound, hypertension should result though the blood

supply to the kidney is not reduced below that required under ordinary circumstances should the work of the kidney be increased, but any corresponding augmentation of renal blood flow prevented. In other words, is a disparity between the work demanded of the kidney and the renal blood flow, rather than any real ischaemia of the renal tissue in a pathological sense, the occasion for the discharge of renin into the circulation? We (Greenwood, Nassim and Taylor) tested this conception in dogs. One kidney was enclosed in a rigid cast made of strips of gauze soaked in collodion. This procedure caused no rise in blood pressure. Seven days later the other kidney was removed. In all cases, except a few in which the cast had failed to act as a rigid encasement for the kidney, a rapid rise in arterial blood pressure followed this second operation. A rise of 50 mm. Hg or more usually occurred within 24 hours. The hypertension appears to be permanent, though no animal has so far been observed for longer than four months. In some instances, retinal haemorrhages and cerebral symptoms supervened.

These results may be interpreted in the following way. It is well known that when the entire renal work is thrown upon one kidney, by the removal of the other, the blood supply to the remaining kidney is increased, a greater number of glomeruli become active and renal hypertrophy results. When these compensatory responses are prevented by a cast, the kidney falls back upon its last resource, namely, an increase in the general blood pressure, brought about by the liberation of renin. Thus the glomerular pressure is increased and, in consequence, the filtration rate.

The experiments of Page should be mentioned here. He found that cellophane wrapped loosely around the kidney set up a reaction which after a time formed a dense perinephric mass of fibrocollagenous tissue. The blood pressure commenced to rise in from 3 to 5 weeks after the wrapping had been applied.

The result of this experiment is not so readily interpreted. At first sight it might seem that the capsule of fibrocollagenous tissue induced by a loose cellophane wrapper acts in essentially the same manner as does a closely applied gauze-collodion cast, the appearance of the hypertension being delayed only because a certain time must elapse for a sufficiently resistant encasement of the kidney to form. However, this cannot be the right explanation for in these experiments no extra burden was thrown upon one kidney, it being found unnecessary to perform unilateral nephrectomy in order for hypertension to develop. This suggests that either the chronic perinephric process leads to constriction of the renal artery or that the vessels within the kidney itself are narrowed by compression. In either event a true ischaemia would be produced. Or the perinephric process may have affected the kidney parenchyma more directly and in such a way as to call into play some quite different hypertensive mechanism. For example, the hypertension of acute nephritis has apparently a nervous, not a chemical basis; it is prevented by denervation of the kidney.

The results of subsequent experiments (Maclachlan and Taylor) designed to bring out the relationship between the hypertensive response and increased kidney work when renal hypertrophy and augmentation of renal blood flow are prevented by means of a cast, have confirmed our belief in such a relationship. Animals showing hypertension caused by the application of a cast to one kidney followed by excision of the other, were kept for a time on a diet of dog biscuit. An extra burden was then thrown upon the kidney by substituting a diet consisting of large quantities of lean meat. A further prompt rise in blood pressure resulted. The first animal experimented upon in this way suffered detachment of both retinas and severe cerebral symptoms; the blood pressure rose from 165 mm. Hg to 242 mm. Hg. Upon returning the animals to the diet of dog biscuit their blood pressures subsided again, though not as a rule to the level existing before they had been put upon the high protein diet. The food was changed several times, each dietary period being of 4 days duration. The pressures in-

variably rose upon the diet of meat and fell again upon changing to biscuits. Urea given by mouth in amounts equal to that calculated as contained in the meat diet exerted the same effect as meat itself.

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A Lesson in English Diction

Executive Mansion,
Washington, Nov. 21st, 1864.

Mrs. Bixby, Boston, Mass.
Dear Madam,—

I have been shown in the files of the War Department, a statement of the Adjutant-General of Massachusetts that you are the mother of five sons who have died gloriously on the field of battle. I feel how weak and fruitless must be any word of mine which should attempt to beguile you from the grief of a loss so overwhelming. But I cannot refrain from tendering you the consolation that may be found in the thanks of the republic they died to save.

I pray that our Heavenly Father may assuage the anguish of your bereavement, and leave you only the cherished memory of the loved and lost, and the solemn pride that must be yours to have laid so costly a sacrifice upon the altar of freedom.

Yours very sincerely and respectfully,

A. Lincoln.

An engrossed copy of this facsimile letter of President Lincoln to Mrs. Bixby, hangs on the walls of Brasenose College, Oxford University, England, as a specimen of the purest English and most elegant diction extant. It is said that as a model of expressive English, it has rarely, if ever, been surpassed.

A PROBLEM IN DIAGNOSIS

SUMMARY OF CLINICAL FINDINGS

By K. M. ALFORD, M.D.

Baby Boy K.P., thirteen months old, male infant, admitted to the Hospital for Sick Children, April 21, 1939, with a temperature of 103 degrees and weight twenty-two pounds, four ounces. He was well until eight weeks prior to admission, when while playing on the floor he suddenly began to choke, became blue and coughed frequently. He was held head downwards, his back was slapped, and he brought up a small amount of mucus and bright red blood. Since then the child has had a persistent, distressing cough. The day following the attack the child's temperature rose to 104 degrees, an X-ray was taken and pneumonia reported. The child was put on sulphapyridine daily for three weeks with little change in condition. The temperature gradually subsided and the child seemed to improve. Thirteen days prior to admission, however, the temperature rose again to 103 degrees. His physician again reported that pneumonia was present and again sulphapyridine was given and continued up to the time of admission. Family history and past history are of no signficance.

Physical Examination

Revealed a fairly well developed, moderately well nourished infant, very pale and acutely ill, with some slight respiratory distress. Pharynx was moderately inflamed with mucopurulent postnasal discharge present.

Glandular System — Discrete, slightly enlarged, cervical, axillary and inguinal glands palpable.

Abdomen—The liver was palpable two finger breadths below the right costal margin and the spleen was felt three finger breadths below the left costal margin.

Chest—Showed diminished movement on the right side and there was dullness to percussion over the entire right lung both anteriorly and posteriorly. Bronchial breathing was heard at the right apex and over the entire lower lobe on the right side depressed breath sounds were present. No rales or rhonchi were heard.

The rest of the physical examination was entirely negative.

Laboratory

The urine was negative, I.C. 1:400 negative. The Hgb. on admission was 30% and following transfusion the R.B.C. was 6,300,000, Hgb. 58%, W.B.C. 14,000, Polys. 53%, Lymphs. 39%, Eos. 2%, Myelos. 6%. Fluid from the bronchus grew hemolytic and non-hemolytic strept., and staph. albus and aureus.

X-ray of chest taken in the upright position showed the right lung to be practically opaque with the heart displaced to the right.

Course

On the first admission the child was bronchoscoped three times and a great deal of foul smelling material was aspirated from the right bronchus. Following this there was a definite and steady improvement and the right chest cleared slowly. Was discharged to the Preventorium on May 19, 1939. He had twelve subsequent admissions coming in from the Preventorium about every two weeks for bronchoscopic suction. He had sixteen bronchoscopic suctions in eight months. Following each suction there would be temporary improvement in the findings in his right chest and the foul-smelling copious sputum would be relieved. His last admission was on December 23, 1939, when he came into the hospital with a history of having had a fever between 101 and 102 degrees for

eight days. During this admission he had two bronchoscopic suctions performed, transfused prior to both suctions, put in an oxygen tent, but in spite of his supportive treatment his temperature ranged between 103 and 104 degrees. He went downhill rapidly and died December 30, 1939.

SUMMARY OF AUTOPSY FINDINGS

By W. L. DONOHUE, M.D.

The post-mortem examination was done 8 hours after death.

The body was that of a well developed and poorly nourished boy of 21 months. The findings of interest were entirely confined to the chest.

The mediastinum was shifted to the right for a distance of approximately 2½ cm. The thymus was small and atrophied but the heart and pericardium were normal except for their lateral displacement. The major portion of the lower and posterior two-thirds of the right pleural cavity was obliterated by dense fibrous adhesions. These were stoutest over the diaphragm. The anterior and upper part of the cavity was patent. The left pleural cavity contained no fluid and was, for the most part, free of adhesions except in its lower and posterior part where there were a few easily broken down plastic fibrinous adhesions. The right lung consisted of only 2 lobes. The middle and upper lobes were joined. This lung was shrunken and considerably smaller than the left. Relatively the lower lobe was much more shrunken than the upper. The pleural surface over the entire lower lobe and the major portion of the posterior aspect of the upper lobe was roughened and shaggy with numerous fibrous adhesions. The lung was, for the most part, a dark mottled purplish red colour. In the anterior and upper part of the lung there were a few areas where the pleura was salmon pink and the lung air-containing. Elsewhere the lung was non air-containing and solid and lumpy to palpation. The cut surface of the lung showed the greater part of both lobes to be honey-combed with large dilated bronchi which contained foul pus. The intervening lung tissue was consolidated and showed an interstitial fibrosis. This was most marked in the lower lobe. In the outer and lower aspect of the lower lobe approximately 0.5 cm. from the pleural surface was a small irregular abscess cavity measuring 1.5 cm. in diameter. This contained pus similar to that encountered in the bronchi, and in addition several small fragments of a foreign material. The largest of these did not measure over 0.5 cm. in diameter.

The left lung was voluminous and emphysematous with irregular patches of consolidation scattered throughout both lobes. In the upper part of the upper lobe and in the lower lateral part of the lower lobe there were small groups of dilated bronchi containing pus similar to that found elsewhere.

The lymph glands about the bifurcation of the trachea and hila of both lungs were markedly enlarged and oedematous, some measuring up to 2 cm. in diameter. This was most marked on the right side.

Investigation of the foreign bodies found revealed them to be portions of the seed bearing head of timothy. One seed was, in fact, found.

The microscopic examination of the lungs showed chronic abscess formation, bronchiectasis in various stages, chronic interstitial fibrosing pneumonia and an acute pneumonic process.

Bacteriological investigation of the lung revealed a mixture of organisms including spirochaetes and fusiform bacilli.

The anatomical diagnosis was:

1. Aspiration of foreign body (head of timothy).
2. Lung abscess.
3. Chronic bronchiectasis.
4. Bronchopneumonia.
5. Chronic lymphadenitis of peritracheal and peribronchial lymph nodes.

DISCUSSION

This infant was perfectly well for the first 10 months of his life, with no history of any previous upper respiratory or infectious disease, when suddenly he developed cyanosis due to a severe attack of coughing and choking. With the history of playing on the floor just prior to such an attack the most probable explanation would be the aspiration of a foreign body. As this patient lived in a rural district, evidently someone coming into the home brought in on their shoes the head of the timothy plant, and while playing on the floor he got this part of the plant in his mouth and subsequently aspirated it into his lung. After this episode, an X-ray was taken which showed what the family physician thought to be pneumonic consolidation, and no foreign body could be seen because of the vegetable nature of the material aspirated. It wasn't until 8 weeks later when he was admitted to hospital that bronchoscopic suction was done and by that time the foreign body was apparently inaccessible, because many bronchoscopics were performed and no foreign body ever seen. The X-ray examinations were of no further assistance in determining a positive diagnosis.

The diagnosis on admission was foreign body in the lung with lung abscess, but because no foreign body was found it was felt by many that this was a lung abscess following what apparently was a pneumonia and the 3 cc. of thick pus which was obtained by thoracentesis during the first admission was probably due to the needle entering the abscess cavity. As the child continued to come in for his bi-monthly bronchoscopic suctions his extremely foul-smelling sputum was indicative of extensive bronchiectasis and this was confirmed by lipiodol injection following one of the bronchoscopic suctions. The lipiodol injection done in October, 1939, showed also some bronchiectasis in the left lung as well as the right.

The patient seemed to be doing nicely on repeated suctions until about 3 months prior to his death when he developed streptococcus pharyngitis and from that time on he went gradually downhill and was never in good enough condition for more drastic treatment such as lobectomy. There is no doubt that all the post-mortem findings in the lungs of this boy resulted from the aspiration of the foreign body.

"Is Tuberculosis Contagious?"

"The contagious nature of consumption has been believed by some authors of high authority, at the head of whom may be placed Morgogni, and altogether disbelieved by others. In the south of Europe the general opinion is in favour of contagion, in the north of Europe against it. As the subject scarcely admits of being confirmed or refuted, and as every medical man in this country has too frequent opportunities of making his own observations and forming his own judgment upon it, it would be profitless to adduce authorities or detail opinion. The view which I take of tuberculous cachenia, without which, in my opinion, tuberculous disease of the lungs cannot occur, leads me entirely to disbelieve that consumption can be communicated by contagion. But I consider the practise of sleeping in the same bed, or even in the same room with a patient in the advanced stage of consumption, highly objectionable, because the rooms of the consumptive are rendered peculiarly injurious to health by the nature of the disease, and the confined atmosphere and high temperature in which they are too often kept.

A Treatise on Pulmonary Consumption
Sir James Clark, Bart., M.D., F.R.S.
Sherwood, Gilbert and Piper
Paternoster-Row, London
1837

LEGG-PERTHES DISEASE

By CHARLES W. TAYLOR, '40

Legg-Perthes disease or coxa plana is a chronic disease of the head of the femur in young children, characterized by limping, limitation of movement, and a strong tendency towards spontaneous cure.

History

It had long been taught that in the common hip joint affection of childhood —tuberculous arthritis—rapid spontaneous healing may occur, with the production of a joint which shows little or no ultimate interference with its function. Thus Brodie, writing in 1834 on scrofulous disease of joints, states: "If the joint receive very early attention, the function of the joint may be wholly unimpaired." In more recent times, Rollier of Leysin, reviewing his cases of tuberculous arthritis of the hip joint treated between the years 1903 and 1913, stated 73 per cent showed restoration of joint function. This conclusion embodied current surgical teaching until the beginning of the last decade.

In 1909, Legg of Boston, in a communication before the American Orthopaedic Association, described a small group of five cases in which were exhibited the symptoms of a mild hip disease with distinctive radiographic appearances. He pointed out that cases of this type had formerly been regarded as examples of benign tuberculous arthritis, but were, in his opinion, a definite clinical entity of non-tuberculous type. He suggested as an explanation of the pathogenesis of

the condition, that a previous trauma had produced an alteration in the vascular supply of the upper end of the femur, and that the characteristic bony changes were the direct result of this disturbance.

In July, 1910, Calvé, of Breck, France, evidently unaware of Legg's work, published a paper containing a detailed consideration of ten cases which had come under his notice in a series of five hundred patients suffering from presumed tuberculous coxitis. These cases differed from ordinary hip-joint tuberculosis, one of the chief features being, as in Legg's

cases, a peculiar radiographic change. Calvé regarded this as an atypical manifestation of rickets, the usual stigmata of which were present in all of his patients.

Independently, in October, 1910, Perthes of Tübingen, Germany, published a careful account of the clinical and radiographic signs of a type of arthritis deformans juvenilis, which proved to be identical with the condition described by Legg and Calveé. In 1913, because of his inability to demonstrate any true arthritic changes, he submitted the term osteochondritis deformans in a paper based upon an observation of twenty-one cases.

Since 1915, there has been a steady addition to the literature on this subject from various countries, but the names of Legg, Calvé and Perthes must remain conspicuously associated with the original recognition of this disease. Some of the more recent literature has shown a polemical flavour as the question of the priority of the original discovery has been raised. Thus Waldenstroem now claims to have described the condition himself in 1909 and further states that the first recognition of the disease dates back to 1898 to a publication of Maydl. But it is to be remembered that a retrospective analysis of any series of hip joint affections in children will always bring to light, examples of coxa plana.

H.D. Aged 7½ years. Male. Admitted to H.S.C. April 26, 1935.

Two weeks prior to his admission, this patient began to complain of pains shooting down his right thigh as far as his knee. They were severe enough to keep him awake at night. One week prior to admission he developed a limp in the right leg. This had become progressively worse, but his pain had almost completely disappeared by the time he was admitted. On examination, there was a marked limp in the right leg, and he walked with the pelvis tilted down on this side, the thigh internally rotated, and the knee flexed. There was no limitation of movement at either the hip or the knee joint. No tenderness was elicitable. Provisional diagnosis was tuberculosis of the hip joint. An intracutaneous tuberculin test was negative to the 1/40 dilution. He was discharged eleven days later, only slightly improved.

He rested at home for six weeks and returned to the out-patient department with scarlet fever. He was sent home where he remained quietly for another six weeks. He then returned to the out-door patient department without any limp or pain and was instructed to resume activity. He remained free from limp and pain for one week only, when both returned. Three weeks later, on October 21, 1935, he was readmitted to hospital with a diagnosis of osteochronditis of the head of the right femur. On examination, he had slight limitation of external rotation. There was ½″ of shortening, the right thigh was 1″ and the calf ¼″ smaller than the left leg. Forcible adduction caused slight pain. He walked on the ball of the foot with the hip and knee both slightly flexed, and with a protective limp, hurrying off the leg.

X-ray examination revealed a generalized increase in density on the affected side, a flattened head with localized areas of increased density giving a fragmented appearance. The joint space was clear and 4 mm. wider than on the corresponding side. The neck was 5 mm. wider, at its narrowest point, shortened by 6 mm., and the angle between it and the shaft of the femur was decreased. It is reproduced above.

He was fitted with a Taylor splint and discharged five weeks later. After wearing this for twenty-three months, he had a truly remarkable result, with no limitation of movement, and a radiological picture which showed almost complete recovery of the affected hip.

Etiology

The usual age of onset is between three and twelve years although the disease is occasionally encountered in adults and in infants. It is three to four times as common in the male as in the female; and, while either hip is equally liable, bilateral cases occur in only one out of every twenty cases. Nebulous and indeterminate is the atmosphere which surrounds the various theories which have been formulated to account for its origin. Chief among the more tenable of these are the infective, vascular, traumatic and developmental theories.

(1) *The Infective Theory*

Platt, the ablest of the brave defenders of this postulate, considers this disease to be the result of a definite infection of low-grade virulence which reaches the femur by the blood stream, and gives rise to changes which are subchondral in location. Clinically, he bases this conclusion upon the occasional association of an infective process (as evidenced by either the history of some definite preceding acute infective illness, such as a mild

polyarthritis) or by the occurrence of coincident signs such as pyrexia and vague ill health. His pathological basis is the histological study of Phemister of Chicago who curretted the interior of an epiphysis from a known case at operation. The contents gave no microbic growth, but the changes demonstrated in the tissue removed were claimed to be typical of an old infective lesion probably of pyogenic origin. At operation, the joint cavity gave evidence of an active synovitis, but the articular surface of the deformed head retained its normal sheen. To offset this meagre and inconclusive evidence of infection, are the observations that the patients with coxa plana are regularly distinguished by their healthy appearance, the small amount of pain, the absence of fever, and the fact that marked X-ray changes are seen in many clinically early stages which is in marked contrast to early infective bone conditions.

(2) *The Vascular Theory*

Zemansky and Lippman contend that the blood supply to the head of the femur is derived from two sources: (i) Perforating vessels from the diaphysis which penetrate the epiphyseal cartilage and supply the base of the caput. (ii) Blood vessels carried by the ligamentum teres femoris, which supply the central region. In infancy, the chief nutrition of the head is derived from these vessels in the ligamentum teres, those coming from the diaphysis being relatively unimportant. As adolescence progresses, these former vessels become of less importance, until the epiphysis unites with the shaft, at which time, they no longer carry blood into the femur, the nutrition of the crest being derived entirely from below. These investigators suggest that the immediate cause of the disease lies in some maladjustment of the delicate physiological balance that must exist between these sources of nutrition to the femoral head. They claim that a series of experiments in rabbits in which the ligamentum teres femoris was ligatured produced a necrosis of the caputs analogous to Legg-Perthes disease in humans.

(3) *The Traumatic Theory*

This was propounded by Legg in his original article. It is supported by the definite history of trauma in many cases, by its preponderance in boys (who are proverbially more traumatized) and by its not infrequent occurrence in cases of congenital dislocation of the hip, particularly if some difficulty has been experienced in reduction. As a result of the injury, there is obliteration of a portion of the vascular supply with consequent atrophy. A compensatory hyperaemia of the femoral neck is the natural response, and is the starting point of those hypertrophic changes which are denoted by the occurrence of broadening, and increased density.

(4) *Congenital Abnormalities of Ossification*

Some investigators, by a process of elimination of the above factors in their own minds, have concluded that the changes in the head of the femur were purely developmental, and indicative of an abnormal delay in the formation of the nucleus of the epiphysis. Against this stands the common appearance of coxa plana in a hip previously known to present a normal femoral head as revealed by radiographs.

Morbid Anatomy

There has been very little opportunity to study the pathology of this disease, as it is never fatal, and never should be operated upon. A marked lack of agreement exists among the few who have had such an opportunity in interpreting what they have seen, and even in the description of their findings. The majority record no change in synovial membrane, normal articular cartilage, and a quiet aseptic necrosis of subchondral bone. Buds of cartilage grow down from the periphery, and after a variable period of time, recalcification and reshaping occur, and the disease subsides, leaving a certain amount of residual deformity.

Symptomatology

This disease has only two symptoms, *viz.* a limp, and pain. The limp is constant, the pain very inconstant.

In the majority of cases, the patient suffers some minor trauma (such as a fall from a verandah) following which he has a slight limp for a few days. This subsides and he is apparently well for approximately three months. Then he begins to limp again. This may be so slight as to be scarcely noticeable. The parents may notice it before the patient himself becomes aware of it. In its earliest stages, it is often better heard than seen. It may occur only in the evening, or after some unusual feat of exertion. To become progressively more marked until it is present all the time, is the usual tendency. On the other hand, it may clear up spontaneously, only to recur again. This may be the only complaint the patient has.

More often, however, there is some accompanying pain. It varies from a slight ache in the region of the joint or thigh at the end of the day, to fairly severe pain constantly present and aggravated by movement. This latter is the less common of the two extremes. Not infrequently, a case is seen in which the pain is referred to the ipsilateral knee-joint. These may suggest disease of this joint and lead to erroneous diagnosis unless the examiner is on the *qui vive* for such cases.

Physical Signs

As in any affection of joints, the limb is subjected to four types of examination —inspection, palpation, movement and measurement.

Inspection. The patient's gait is first noted. Except in those cases in which there is much pain, the child does not gingerly hurry off the affected side. Instead of favouring the joint, the full body weight is borne upon it. On the other hand, the character of the limp suggests that there is limitation of movement. There may be a slight flexion deformity so that the heel is not placed on the ground, the step being taken on the ball of the foot.

On exposing the region, it does not appear abnormal. There is no swelling, redness, or other earmark of inflammation, either acute or chronic. The only indication of pathological change may be slight atrophy. Usually this is so slight as to escape detection on inspection.

Palpation. There is no difference in temperature between the two sides. Usually no tenderness is demonstrable. When present, it is deep-seated and in the immediate neighbourhood of the joint. Consequently, it requires considerable pressure to elicit it.

Movement. On testing flexion, extension, abduction, adduction, internal and external rotation, there may be no limitation whatever. More often careful examination will reveal slight limitation of all movements. Marked limitation of abduction and internal rotation with moderate limitation of flexion are often encountered. Occasionally all six movements are markedly impaired. This restriction is largely due to spasm of the muscles. This spasm may be so severe as to completely fix the joint. When such fixation is present, the position is often one of slight flexion combined with adduction. On resting in bed for two or three days, this disappears leaving some residual limitation due to the deformity of the head.

Measurement. It is usual to find some atrophy of the leg muscles and in some few cases actual shortening is present. The atrophy of the thigh may be as much as 1½″; in the calf, not more than ¼″ is encountered as a rule. In advanced cases, with flattening of the femoral caput, shortening up to ⅜″ may be found.

Radiological Examination. This is of the utmost importance, as without it, a diagnosis cannot be made.

The earliest change consists of rarefaction of bone on the diaphyseal side of the epiphyseal plate extending in a band across the neck of the femur. It is thought that this is due to the interference with the blood supply of the region. As the disease progresses, the head becomes involved and shows a peculiar combination of atrophy and increased

density. Atrophy of the periphery of the head occurs with an increase of density in localized areas in the centre of the head, the combination of which produces a fragmented appearance. Coincidently, flattening of the head is seen which varies all the way from a slight diminution of the normal rounded contour to an irregular disc which sits on top of the neck of the femur and overlaps it to some extent. The contour becomes somewhat irregular in outline, as does the acetabular margin in the later stages. The joint space widens slightly due to an increase in the thickness of the articular cartilage, but remains perfectly clear—a valuable diagnostic point in the differentiation from infective conditions. The neck of the femur is often increased in density, and of greater diameter than that on the normal side. There is a distinct tendency for a diminution in the angle between the neck and the shaft of the femur to occur. Normally this angle is about 125°. In coxa plana, this may be decreased by 5 to 8 degrees.

Diagnosis

A child between the ages of three and twelve years, complaining of a limp or pain in the hip, thigh or knee, may have coxa plana. The hip should be carefully examined for limitation of movement, particularly internal rotation and abduction, and for spasm and pain at the limits of motion. The presence of these findings even in slight degrees, indicates the need for a roentgenographic examination, by which the diagnosis can almost always be established. In questionable cases, the patients should be carefully observed, their activities should be limited, and repeated roentgenographic examinations should be made.

The X-ray and other features of this condition are so different from those of other conditions of the hip occurring in this age period—fracture, dislocation, osteomyelitis, suppurative arthritis, acute rheumatic fever, and bone tumours—that differential diagnosis should present little difficulty after a careful and complete examination has been made. Tuberculosis of the hip is the comonest and gravest affection, necessitating early diagnosis and treatment if the functional utility of the joint is to be restored. It must therefore be first excluded. In mild tuberculous arthritis, the symptoms and signs may be identical, and the diagnosis can only be made on X-ray examination where thinning of the joint space, atrophy and decalcification, lack of broadening of the neck and head and later, destruction at the surface of bone are seen. The intracutaneous tuberculin skin test is of value when negative, as under this circumstance, the weight of evidence is decidedly against tuberculosis.

Course in Untreated Cases

When no treatment is used and the disease is allowed to follow its natural course, that course is usually somewhat as follows: The onset may be sudden or gradual and is accompanied by a limp or pain which is usually slight at first but increases as the disease progresses. Disability is variable and is usually proportional to the severity of the symptoms. At this time, there is limitation of movement with pain and spasm on motion. Radiographically, the femoral head is flat and irregularly increased in density and the joint space is wide but clear. After a variable period, usually six to twelve months, the symptoms and signs gradually subside, over a period of one to two years, although the limp and slight limitation of motion may persist indefinitely. In the meantime, the roentgenogram of the femoral head reveals the disapperance of the increased density, and in its place irregular ossification, which gradually returns to normal texture. The flattening of the head and widening of the joint space may largely disappear, but the head is more likely to have become permanently deformed.

Treatment

The first step in treatment is to establish the diagnosis. Once this is done, the patient should be confined to bed for a period of two to three weeks. This relieves the pain, and the muscular spasm subsides, so that almost a full range of

movement may be obtained at the end of this time. If pain be severe, a Buck's extension should be applied to the limb for this period, with a tension of about ten pounds for an average eight-year-old boy. The foot of the bed is elevated to counteract this weight.

During his stay in bed, the patient is measured for a Taylor walking splint, and this is constructed to suit the individual requirement. In its essence, it consists of a splint for the affected limb which extends below the heel for two inches and up the side to below the costal margin in the short variety, or to below the axilla in the long variety. In these locations, a girdle encircles the body. The weight is transferred from the splint to two straps which pass beneath the perineum and are attached to the girdles. Thus the weight of the body is not carried by the affected limb, but by the perineum. A two-inch cork lift is put on the boot of the sound side to compensate for the distance the opposite heel is raised off the ground by the splint. The patient is allowed up and about in this splint for a period of from two to three years, until radiographs show the lesion to be well healed. Activities which would cause a severe wrench or trauma to the hip—such as jumping from a height, skating or playing football—should be prohibited when the splint is discarded.

Results of Treatment

If treatment does not shorten the course or diminish the severity of the disease and its sequelae symptomatically, objectively, or anatomically, it has failed. The type of treatment outlined accomplishes this result more thoroughly than any yet devised. The use of casts is strongly advised against as they encourage subsequent limitation of motion. The various operations devised for drilling or curetting the head, are as yet experimental and not of proven value. Their rational is very questionable. They are technically difficult and there is every danger of serious damage to the hip. The simple and more conservative treatment outlined, yields excellent results. And as we have often heard before, "You can't beat results". The functional results are almost 100 per cent, and radiographic examination of healed cases shows an amazing degree of return to normality in even some of the initially hopeless looking cases. A very flattened fragmented head will regain its normal uniform density and natural contour to such an extent that close inspection is required to determine which is the affected hip.

Summary

Legg-Perthes disease occurs chiefly in children between the ages of three and twelve years, more often in boys than in girls. It is probably caused by some slight trauma which interferes with the blood supply to the head of the femur, possibly by causing a slight slipping of the head on the neck of the femur. It is characterized by a limp, and limitation of movement, due to muscular spasm. The characteristic radiographic changes which consist of atrophy of bone on the diaphyseal side of the epiphyseal plate, flattening and "fragmentation" of the head, widening of, but a clear joint space, with increased density and thickness of the femoral neck, slight coxa vara, all distinguish it from tuberculosis of the hip joint. It tends to clear up spontaneously, but by a short period of rest in bed followed by the use of the Taylor hip splint for upwards of two years, a better anatomical and functional result is obtained in a shorter time.

The writer wishes to thank Dr. J. L. McDonald for his helpful criticism and suggestions, and Dr. A. H. Rolph for his kindness in furnishing the radiograph.

An extensive bibliography will be supplied upon request.

IDIOPATHIC ULCERATIVE COLITIS

By N. LITTNER, '40

Idiopathic Ulcerative Colitis is an ulcerative inflammatory condition of the rectum and colon of unknown aetiology, which is characterized by a chronic course with numerous remissions and relapses.

This disease has also been called "Chronic Ulcerative Colitis", "Colitis Ulcerosa Gravis" and "Non-Specific Ulcerative Colitis".

Incidence

Frequency — Uncommon; Buie[1] reports it as occurring in 2.6% of all proctologic disorders among patients at the Mayo Clinic, while only 0.38% of all autopsy cases at the Banting Institute were due to this cause.

Sex—Probably of the same frequency in both male and female, although some authorities report it as being twice as common in females, while others report the reverse. The sex does not seem to influence in any way the age of onset, the type, or the severity of the disease.

Age—This is a disease of early adult life, occurring chiefly in the 20-40 age group. However, cases have been reported between the extremes of two years and eighty years.

Occupation—Most of the patients are sedentary rather than manual workers, and are of a higher educational standard than the average hospital class.

Race—Cases have been found in practically every racial group.

Climate—There may be a seasonal (spring and fall) incidence of attacks. Bargen believes that the cases may occur more frequently in a climate which predisposes to upper respiratory tract infections.

Geography—There is a universal geographic distribution. Buie has suggested that variations in the same disease in different countries, because of changing customs and habits, may be responsible for the startlingly diverse opinions put forth, as to the origin of the disease, by equally competent workers.

Aetiology

Idiopathic Ulcerative Colitis was first discovered, at post-mortem, by Samuel Wilks in 1875. It was described by Allchin in 1885, and by Hale-White in 1888. It was probably known to Sydenham who, in 1669, described a condition which he called "the bloody flux".

Despite this relatively long period that it has been under investigation, the cause of Idiopathic Ulcerative Colitis, as its name suggests, is as yet unknown. However, numerous theories have been suggested:

(A) Infection

(1) *Locally Infective*

(a) *B. Dysenteriae*—Hurst[2] has long considered the disease to be the result of continued infection by the dysentery bacillus.

(b) *Bargen's diplococcus*—Bargen,[3] in 1925, isolated a lancet-shaped, gram-positive diplococcus from cases of Idiopathic Ulcerative Colitis. Since then, he and his associates have claimed that this bacterium is responsible for, and can be isolated in 80% of, all cases of the disease.

(c) *Bacterium necrophorum* — Dack[4] and his colleagues recently have suggested an anaerobic bacterium as the primary infective agent.

(d) In like fashion, most of the organisms from the human colon, such as the colon bacillus, the pneumococcus, an organism resembling Bacteroides fragilis and funduliformis, actinomyces necrophorus, enterococci, streptococci and even viruses, have, at one time or another, been suggested as important aetiological factors.

(2) *Focally Infective*

As in other diseases of obscure origin, the question of focal sepsis has been re-

peatedly raised. However, most authors agree that focal sepsis is more important in causing exacerbations of the condition rather than acting as a primary aetiological agent.

(B) **Dietary**

(3) *Primary Dietetic*

Anderson[5] was the first to advance the idea that food allergy might play a primary role in certain cases of Idiopathic Ulcerative Colitis. He believes that sensitization by, followed by exposure to, the specific antigen in the food produces an allergic inflammation which may be accompanied by actual necrosis of the bowel mucous membrane.

Such foods as milk, egg, orange, wheat, spinach and tomato have been suggested —in the order named—as a primary, as well as an exciting, factor in the disease.

(4) *Secondary Dietetic*

Mackie,[6] as well as many others, believes that secondary deficiency (the deficiency, especially of vitamins, being due to the continual diarrhoea which is a feature of this condition) constitutes an essential part of the underlying mechanism.

(5) *Decreased Gastric Acid*

Achlorhydria, or subacidity of the gastric juice—it is claimed—is present in most cases and is of some importance in the aetiology of the disease. However, Cullinan and others have disputed this finding.

(C) **Psychogenic**

(6) Sullivan[7] believes that about 75% of cases of Idiopathic Ulcerative Colitis are of psychogenic origin.

Wittkower,[8] in a recent study of 40 cases, found that 37 of them showed emotional disturbances superimposed on an unusual or abnormal personality. No uniform personality type could be established, although obsessional and hysteric cases were prominent. He suggests that Idiopathic Ulcerative Colitis is "a disease of the mentally ill or maladjusted".

(D) **Constitutional**

(7) The refuge of hereditary weakness of the bowel wall has been adopted by some authors. However, few figures on family incidence have been recorded, and this is still, therefore, an open question.

The evidence so far available does not justify us in saying that any one of the above named factors is *the* cause of Idiopathic Ulcerative Colitis. That *infection* is frequently present cannot be denied, but it is yet to be conclusively proven that it is other than a secondary invader of ulcers previously formed. *Sensitization* of the colon to foreign proteins of either bacterial or dietary origin may in some cases play an important role in the mechanism of relapse and activity of the disease. It is probable that *secondary deficiency states* not infrequently occur and, occasionally, may even assume major clinical importance. *Psychological disorders*, by adversely affecting the secretory and motor physiology of the gastrointestinal tract, may also be of considerable importance. However, it must be emphasized that, despite considerable investigation, the cause of the condition is unknown.

Pathology

(a) *Course of the Disease*

According to Buie there is an initial hyperemia of the superficial mucous membrane with edema and thickening. This is followed by the formation of numerous minute miliary abscesses in the mucous membrane. When these rupture, leaving myriads of miliary ulcers there is found the typical picture of the disease—the one usually seen by the physicians — the "granular moth-eaten bleeding mucosa."

During a remission the miliary ulcers heal leaving pock-like scars present in the bowel wall.

Secondary infection of the ulcers results in deep, jagged ulceration which is most severe in the rectum and which tends to overlie the tineal bands of muscle fibres in the colon.

Repeated attacks of this deep ulceration with subsequent fibrosis, results in thick-

ening of the bowel wall, contraction of the lumen, and occasional stricture at an area of severe localized ulceration.

(b) *Distribution of the Lesions*

The lesions begin in the rectum in 95% of cases and extend up to involve the entire colon in 60% of cases. Late in the disease the terminal end of the ileum may become moderately inflamed. arely, the disease is localized to only part of the large bowel.

(c) *Complications*

There are numerous complications found, most of them being due to chronic diarrhoea with loss of many required food elements and the presence of an infective focus.

(i) Bed sores, (ii) polyposis of the colon, (iii) stricture of the large bowel, (iv) perirectal abscess, (v) perforation of the colon, (vi) cutaneous lesions, and (vii) a rheumatoid form of arthritis, are among the more common.

Symptoms

(A) **Onset**

The onset is usually fairly sudden, the first symptom being an increasing frequency of bowel movement (the stools remaining formed), and the passage of blood and mucus. This initial illness is seldom acute, fever being uncommon. The general health usually remains surprisingly good for some time, so that the patient does not usually seek advice from a physician until after a month or more has passed. Frequently, a patient first comes in during an attack of severe diarrhoea with liquid stools, and only on close questioning is the above history of onset elicited.

Although an attack of frequent bowel movement and blood is the most common onset, constipation and blood passage, or even normal bowel action (although accompanied by blood) may occur.

This first attack may only last several weeks; occasionally, it lasts years.

(B) **Types**

(1) *Moderately severe, recurring type* comprises the great majority of cases. The disease pursues a chronic course lasting many years, and is marked by a succession of relapses and remissions.

During a *remission* the patient may feel perfectly well and go on with his usual work.

Relapses may be brought on by (1) emotional factors, such as shock, worry, anxiety, fear or embarrassment, (2) an acute infection, especially upper respiratory, (3) dietary indiscretion or food poisoning, (4) change in weather, especially exposure to cold and dampness, and (5) unknown causes.

Severe attacks may occur at any time following the initial illness. The patient may become gradually, or quite suddenly, extremely ill. Nervous symptoms, such as marked prostration, depression and anxiety, may occur. Flatulence, a feeling of abdominal distension, loss of appetite, nausea and even vomiting, mild lower abdominal crampy pain which is relieved by defecation, and diarrhoea with soft or watery stools, blood, mucus and frequently pus is the typical picture.

The bowels may move anywhere from 5 to 50 times daily. Fever may be continuous or intermittent, during a severe attack, and is always accompanied by a change in the patient's condition for the worse.

After an illness lasting, perhaps for months, and during which the patient may have become almost moribund, there is frequently a remission with recovery. The profound loss of weight and strength during a severe attack is one of the outstanding features of the disease; yet, there are few diseases in medicine where the patient may reach such a stage of exhaustion and emaciation and then make so spectacular a recovery—with or without treatment.

(2) *Mild Type* may go on for 10, 15 or 20 years with little or no inconvenience, and the patient able to carry on his or her daily duties.

(3) *Acute fulminating type* occurs in about 5% of cases. The onset may be exceedingly severe with a septic course, bouts of fever and severe bloody diarrhoea. The mortality is very high.

Signs

(1) Physical Examination

(a) *General:* If in a severe attack, patient will appear pale, severely emaciated, extremely ill and the temperature may be as high as 103° F.

(b) *Abdomen:* The abdominal wall is often thin. Although there may be a sense of fullness on palpation, visible distension is rarely seen. Occasionally, there is retraction.

Rigidity is rare. Tenderness, when present, is mostly in the lower part of the abdomen and along the course of the large bowel, particularly over the descending colon.

The caecum and descending colon are often palpable, while the latter sometimes feels contracted and mobile.

(c) *Blood-pressure:* During the severe phase the blood-pressure is often low and the systolic pressure may be under 100 mm. Hg.

(d) *Rectal examination:* The sphincter is often contracted and digital examination painful to the patient. The mucous membrane may feel velvety in the early stages of the disease and rough in the late stages. There may be blood on the examining finger. Frequently, rectal examination is completely negative.

(2) Clinical Tests

(a) *Stools:* May be well-formed in the early stages of the disease, with blood well-mixed with the stools (probably from the colon) or on the outside of the stools (probably from the rectum). As the disease progresses, the stools become offensive, frequent, fluid, thin and soap-like, consisting of fecal material closely admixed with blood, mucus and often pus (evidence of ulceration).

If the lesion is confined to the recto-sigmoid region, blood and lumps of blood-stained mucus and also pus may be passed many times a day either separate from feces or without any fecal material at all.

Parasites and dysenteric organisms are not usually found.

(b) *Blood:* (i) Most cases show a moderate to severe anemia which becomes more marked during a relapse and recovers again with a remission. Thus the hemoglobin may fall to 20% during a severe attack, yet rise to 90% in the following recovery. This anemia may be either an iron-deficiency anemia (C.I.= 0.5) due to haemorrhage, or a normocytic anemia (C.I.=1.0) because of secondary infection of the ulcers in the colon with resulting toxemia.

(ii) The total leucocyte count or even differential counts may reveal little change from the normal, even during a severe attack with fever. According to Vickers, a reliable index of the degree of toxemia is the great predominance of neutrophilic non-filamented forms (nucleus not segmented).

(iii) An eosinophilia may occasionally be found.

(c) *Sedimentation Rate:* This may be high, but not as high as would be expected if the disease were a primary infection. To obtain accurate figures, allowance must be made for both the anemia and the decreased amount of fluid.

(d) *Agglutination Tests:* Agglutinins to various organisms, including dysenteric bacteria, may be found in low and insignificant titre in the blood. It is possible that they are produced by secondary invasion of the ulcers in the colon.

(3) Special Investigations

(a) *Proctoscopic Examination.* The mucous membrane of the upper part of the rectum and lower part of the sigmoid has a reddened, granular appearance and bleeds easily wherever the instrument touches it.

Early, there may be no visible ulceration. The mucous membrane then becomes more oedematous and superficial ulcers appear, these ulcers being separated from each other by reddened, granular mucous membrane. The ulcers at first are tiny, but later run together to form larger ulcers, often covered with a muco-purulent exudate.

In more chronic cases, there are callous

ulcers with oedematous mucous membrane between them.

During remissions, healing ulcers may be observed, sometimes with normal intervening mucous membrane. In some cases, linear or three-pointed scars of healed ulcers may be found. In long-standing cases rectal strictures may be observed.

Pseudo-polyps are frequently seen. Blood and pus may be noticed coming down from higher in the colon. Sometimes the mucous membrane has a "cobblestone" appearance.

(b) *Barium Enema with X-ray:* In mild cases or in acute fulminating cases there may be no early demonstrable X-ray changes.

Some observers claim that quick passage of the barium around the colon is one of the earliest signs; others, however, have found a hypomotility with prolonged delay of forward passage of barium in the proximal half of the bowel.

A characteristic and frequently the earliest recognizable feature is the complete lack of haustration seen in the distal part, and sometimes in the whole, of the colon, giving it a tubular appearance.

In the moderately severe recurring type, a fine, granular mottling of the bowel shadow, with occasionally a moth-eaten, marbled effect is very characteristic.

In very chonic cases, the full X-ray picture of narrowing, shortening, rigidity, lack of haustrations, extreme irritability and irregular mucosal outlines may be observed.

X-ray findings are very unreliable in this disease and are not as important as proctoscopic examination as a means of diagnosis.

(c) *Fractional Gastric Test-Meal:* Varying degrees of subacidity of the gastric juice as well as achlorhydria have been reported by some authors.

Diagnosis

Idiopathic Ulcerative Colitis is an extremely serious disease with a high mortality rate. It is therefore essential that a correct diagnosis be early established to distinguish it especially from Mucous Colitis, Amoebiasis and Bacillary Dysentery. This is possible by correlating the facts derived from (1) history, (2) laboratory examination of the stool for blood and pus, and (3) proctoscopic examination. If ulcers or a typical granular appearance is not observable through the latter means the diagnosis should be in doubt, because more than 95% of cases of Idiopathic Ulcerative Colitis involve the rectum.

The presence of blood in the stool is to be judged, not on the patient's story, but by repeated laboratory examinations. An occasional slightly positive benzidine test is of no significance, because this may occur in normal stools.

Prognosis

Idiopathic Ulcerative Colitis is a severe, chronic disease with a mortality of 10-40% and no known specific cure.

The duration of life is, on an average, from 6 to 15 years, after the first onset of the illness. Fifty per cent of the deaths occur within 2 years of onset, and 25% more within the next 2 years, the patients usually dying of inanition, toxemia or peritonitis.

Patients may live only 2 weeks or may last 40 years. Complete recovery with no liability to future breakdown is so uncommon that Mackie has suggested that the phrase "apparently arrested" be substituted for the word "cure" in connection with this disease.

Remissions occur in about 30% of cases. The length of the remission is quite irregular and frequently bears no apparent relationship to the number or severity of previous relapses. It is almost impossible to say that, after any given period of freedom from activity, the disease will not recur.

The age of onset seems to have little effect on the prognosis. However, some authorities claim that the highest mortality and lowest rate of improvement are seen in those patients giving a history of symptoms of less than 1 year duration.

It should also be noted that the extent of X-ray involvement is *not* a reliable prognostic sign.

Treatment

The treatment of Idiopathic Ulcerative Colitis is now predominantly medical and surgery should not be attempted (apart from certain specific indications) until all other efforts have proved futile.

Nearly all patients with Idiopathic Ulcerative Colitis will respond well to careful treatment, even when they appear almost moribund. No specific therapy has yet been devised. Moreover, it should be remembered that the reported success of any particular remedy is not necessarily proof of its specificity, because of (1) tendency to remissions, (2) possibility of suggestion playing a large part.

(A) **Medical**

(1) *Rest*

(a) Physical—Complete bed rest during an attack. The patient must remain in bed until all signs of activity (fever, anemia, pus and blood in stools) have cleared up. This may be weeks, months (usually) or years.

(b) Mental—Peace of mind is vital for recovery. The patient must learn to accept his prolonged rest in bed with a philosophical attitude. Worry, restlessness or mental fatigue on the patient's part will not only delay his recovery but may hasten his death.

(2) *Diet*

(a) A simple bland diet during the acute attack, followed by a nourishing, high caloric, low residue diet is important. Much care must be exercised in planning the diet for these patients, because of the need—due to the inadequate absorption and loss of food by diarrhoea—of a far better diet than that of the normal individual.

(b) Prevention of secondary deficiency states is important. The addition of extra vitamins and minerals to the diet is of use in this respect.

(c) The possibility of food allergy should always be kept in mind, and, if suspected, test diet methods may be employed to discover the offending food.

(d) If achlorhydria is present, diluted hydrochloric acid, 30 minims in orange juice, may be sipped during the meal.

(3) A host of other treatments have been suggested, including:

(a) Iron may be given where the anemia is due to chronic haemorrhage.
(b) Sedatives such as phenobarbital, may be useful.
(c) Atropine or Tincture of Belladonna may be tried where there is spasm of the colon.
(d) Adsorbents, such as charcoal, Kaolin or lacteal, have been suggested where the stools are offensive.
(e) Liquid Paraffin may be used indefinitely in those cases where constipation is present. Salines, by mouth, may also be used to keep the stools soft.
(f) Bismuth or Opium (used discretely) may be used in attempting to control the diarrhoea. However, these are not usually very successful.
(g) Neo-prontosil has been found of value by some workers in treating early cases with mild to moderate involvement of the bowel.
(h) Liver extract intramuscularly, in large doses, was found by Cheney to bring about remissions. He believes that liver (he used reticulogen) contains some as yet unknown substance which promotes healing of the bowel together with restoration of normal function and relief of symptoms of the disease. He does not believe that this substance is either Vitamin B-1 or Vitamin B-2 or the anti-anaemic fraction of G of Cohn.

Other Therapeutic Measures

(a) Autogenous vaccines and sera, especially those against Bargen's Coccus, have been extensively used.
(b) Bacteriophage has been tried.
(c) Removal of foci of infection is practically always done.
(d) Saline wash-outs of the bowl, especially when there is much pus in the stools, is recommended by some authorities.
(e) Blood transfusions may be used in emergencies, depending upon the condition of the patient.
(f) Raw Apple diet is sometimes used.
(g) Cod-Liver Oil retention enemata have been suggested.
(h) Strychnine or glycero-phosphates, if tonics are needed.

(B) **Surgical**

Previous to 1925, approximately 50% of patients with Idiopathic Ulcerative Colitis were treated surgically, with a total 5-year death rate of 75%, of whom 45% died post-operatively.

Since 1925, the pendulum has swung

the other way and it is now fairly generally believed that uncomplicated Idiopathic Ulcerative Colitis is a medical problem, with surgery being reserved for certain definite types of cases.

When surgery is undertaken, it should only be after adequate and prolonged preoperative preparation and rehabilitation of the patient.

(1) *Indications*

(a) stricture,

(b) polyposis or neoplasm,

(c) peri-rectal abscess,

(d) right-sided Idiopathic Ulcerative Colitis,

(e) those cases where the large bowel has lost its function and is only serving as a focus of toxic absorption.

(2) *Types*—Elective surgery usually is for the purpose of:

(a) Creation of an artificial stoma proximal to the disease area; this may be an ileostomy, caecostomy or appendicostomy.

(b) Resection of the diseased tissue; this is usually a complete or partial colectomy. It should always be carried out in multiple stages.

It should be mentioned that laparotomy for the closure of a perforated ulcer or for drainage of the peritoneal cavity is practically worthless. Moreover, surgery in the acute fulminating or haemorrhagic cases has few advantages.

SUMMARY

(1) The aetiology of Idiopathic Ulcerative Colitis is unknown. Bacterial, dietetic, psychogenic and constitutional theories of origin have been put forward.

(2) Idiopathic Ulcerative Colitis, although not common, is a very serious chronic disease and an early, accurate diagnosis is very important. This may be accomplished by (a) history, (b) examination of the stools, and (c) proctoscopic examination.

(3) There is no known specific therapy for the disease. The great variety of measures advocated bears mute but impressive testimony to the inadequacy of all.

(4) Rest in bed, psychological education of the patient, and a proper diet are in general use for treatment of the acute attacks.

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The author wishes to express his sincere appreciation of the helpful constructive criticism of Dr. R. F. Farquharson and Dr. E. J. Maltby in the preparation of this article.

He Took No Chances

The doctor examined him twice a year,
He wore his rubbers when it rained,
He slept with the windows open,
He stuck to a diet with plenty of fresh vegetables,
He relinquished his tonsils and traded several wornout glands,
He played golf—but never more than eighteen holes at a time,
He never smoked, drank, or lost his temper,
He did his daily dozen,
He was all set to live to be a hundred,
The funeral will be next Wednesday,
He forgot to stop, look and listen at level crossings.

ACUTE CATARRHAL JAUNDICE

By L. M. HAMPSON, '40

Acute catarrhal jaundice is an acute hepatic inflammation of unknown etiology characterized by jaundice and frequently by gastric symptoms and rejoicing in a long list of synonyms among the most popular of which in this country are acute hepatitis, acute infectious hepatitis, acute infectious jaundice and epidemic jaundice. It may occur sporadically or in epidemics.

Epidemics of jaundice have been recorded since ancient times and have occurred at one time or another in every country in the world. No attempt at classification was made until Weil (1886) published an account of the symptoms in a number of severe cases. Cockayne (1912) went a step further and differentiated between Weil's disease and the milder type by styling the former "infectious jaundice" and the latter "epidemic infectious jaundice". Hurst and Simpson (1934) further subdivide the "epidemic infectious jaundice" into the infective hepatic and the epidemic catarrhal types.

Etiology

No causative organism of catarrhal jaundice, sporadic or epidemic, has ever been isolated. Some regard it as a mild spirochaetosis. Boyd states "cholangitis may occasionally be a cause of transient jaundice. It is probable that the cases of epidemic catarrhal jaundice are of this nature possibly due to the paratyphoid group of bacteria."

The old view that catarrhal jaundice is due to an ascending infection of the ducts from gastro-duodenitis falls more and more into disfavour. The alternative hypothesis is that it is an hepatitis due to some unknown infection occurring sporadically or in epidemics.

Incidence

An idea of the frequency of the catarrhal type is gained by an analysis of an unselected group of 46 of the most recent admissions of icteric patients to the wards of the Toronto General Hospital. Final diagnosis of acute catarrhal jaundice was made in 17 of this number or approximately 37%.

The disease is almost equally divided between the sexes and almost 50% were under 30 years of age.

Symptoms and Signs

Like so many gastro-intestinal diseases, initial symptoms are of a vague and general nature. For several days before definite symptoms develop, the complaints are of headache with perhaps nausea, loss of appetite and a feeling of lassitude and sleepiness. In 10 of the above 17 cases, a feeling of malaise was the first symptom; in a few, upper abdominal pain and in only two cases did jaundice initiate the train of symptoms.

Vomiting is the first definite symptom. Chills and feverishness may be coincident findings.

The symptoms frequently clear up to some extent until the onset of jaundice 1-14 days later, when a remission of the gastro-intestinal phenomena sets in. In the series under discussion, the interval between onset of symptoms and appearance of jaundice could be determined in 15 cases and the average time was found to be 9 days, varying from 0-14.

Accompanying the jaundice, the patient usually notices the darkness of his urine and the pale quality of the stool, although an occasional bile stool is observed.

Itching, according to Lisney, occurs in those that have the worst jaundice, but this was complained of by only one of the 17 and she had a van den Bergh of only 3.2, while others with van den Berghs running as high as 22.5, 30.0 and 53.0 were free of this distressing complaint.

Physical Signs

Here are found bradycardia, lowering of the blood pressure, sometimes enlargement of the spleen.

The most helpful diagnostic sign is enlargement of the liver which is consistently present to the the extent of one to three or four fingers breadth below the costal margin. In very severe cases the lower edge has been observed to reach the level of the umbilicus. Tenderness of the liver is variable, sometimes even in marked enlargement and severely jaundiced patients being either not present at all or of only a moderate character.

The jaundice is the outstanding feature. According to Meakins the van den Bergh reaction in the earlier stages is indirect, rapidly becoming biphasic and soon a strong direct reaction occurs, these changes indicating the course of the hepatic lesion.

The stools may or may not be pigmented. This would seem to depend on whether the catarrhal lesion is extensive enough to involve both hepatic or common bile ducts.

Laboratory Procedures

The combined urobilinogen and galactose tolerance tests are probably of the greatest value in diagnosing catarrhal jaundice.

The galactose tolerance test was devised by Bauer in 1906 as a test of hepatic function and two years later he reported excessive galactosuria in cases of catarrhal jaundice in contra-distinction to slight galactosuria in cases of jaundice due to obstruction. The test has been used extensively in European clinics but it is only recently that clinical application has been made in this country.

Galactose, after its absorption from the intestine, is carried by the portal circulation to the liver where it is utilized in amounts ranging from 37 to 40 gms. Failing utilization by the liver the galactose reaches the general circulation where it is practically unavailable as a source of energy for any of the other tissues; further, there is no renal threshold for galactose. Thus there is complete elimination in the urine of any galactose which fails to be utilized by the liver, thus justifying the use of the urinary excretion as a measure of the utilization by the liver.

The test is carried out according to the following:

After an overnight fast the patient is instructed to empty his bladder, the specimen being used as a control for glycosuria. He is then given 40 gms. of pure galactose dissolved in 500 c.c. of water with a few drops of lemon juice. Hourly specimens of urine are obtained for 5 hours and in separate containers. During the test period only water is ingested. Each is tested for sugar by the Benedict qualitative test. Those with a positive are mixed and a total sugar determined by the Benedict quantitative reaction.

From the tolerance figures it is seen that a normal individual may excrete from 0-3 gms. of galactose after ingestion of 40 gms. However, when the liver cells are acutely and diffusely damaged as in a toxic or infectious process, the carbohydrate function of the liver is impaired and as a result, amounts of galactose greater than 3 gms. are excreted in the urine in the 5 hours following ingestion of 40 gms. of that sugar.

Since this type of liver cell damage is frequently accompanied by jaundice, the test finds its greatest usefulness in differentiating the toxic and infectious jaundice from the obstructive and hemolytic types.

The use of the urobilinogen test in clinical jaundice is based on the generally accepted view that urobilinogen is formed from bilirubin by the action of certain putrefactive bacteria of the intestine and is eliminated in part, through the kidneys. The disappearance or absence of urobilinogen from the urine in obstructive jaundice has frequently been noted.

It has been observed that excessive amounts of urobilinogen are present in the urine during the beginning and at the end of catarrhal jaundice, and only traces or a total absence during the height of the jaundice when very little, if any, bile is being excreted into the intestine.

From their studies, Wallace and Diamond concluded that the presence of

urobilinogen in the urine in the presence of jaundice points to an intra-hepatic jaundice while its constant absence bespeaks an obstruction.

The Ehrlich aldehyde reaction was chosen for detection of urobilinogen (1.0 c.c. of Ehrlich's aldehyde reagent to 10-15 c.c. of urine). If positive, a red colour appears within 3-5 minutes. The presence of acetone must be eliminated to prevent pseudo-positive reactions.

Course

The course of the disease is usually benign, symptoms soon disappearing. Jaundice lasts 2-8 weeks usually, with an average of 30 days. Mild cases of short duration are not unusual particularly in epidemics.

Pathology

The older view considered the disease a local one, involving the duodenum and lower part of the common duct.

The modern view based not only on biopsy specimens but on tests of liver function would indicate that it is a lesion of the liver parenchyma as well as of the biliary tract.

There has been found degeneration and necrotic changes in the liver lobules as well as an exudative process. The mucous membrane of the biliary tract is swollen with desquamation of the epithelium and exudation of leucocytes. This varies in extent.

Diagnosis

This is rarely difficult in young people. History of gastric symptoms preceding the jaundice by several days, occasional bile-stained stools, enlargement of the liver and the sugar tolerance and urobilinogen tests all serve rapidly to establish the correct diagnosis.

When it occurs after 40 years of age, one is confronted with the additional possibility of cholelithiasis, carcinoma of the biliary tract or head of the pancreas. If there is a reasonable doubt, early surgical inspection is indicated, according to Meakins, since then if it be not hepatitis the maximum amount of benefit may be accomplished either by removal of the obstructing calculus or an early cancer; or if this is impossible, a cholecystenterostomy may mitigate the distressing symptoms of a progressive jaundice.

Treatment

On the whole, symptomatic treatment is carried out. A high carbohydrate diet such as 40 gms. of protein; 80 of fat; 250, sometimes 450, of carbohydrate, is generally prescribed in this hospital.

Summary

Acute catarrhal jaundice may occur sporadically or in epidemics. It is essentially a benign condition, the jaundice clearing up usually in the course of a month. Recognition of it depends on (1) history of gastric symptoms preceding the jaundice, (2) liver enlargement, (3) tests for liver function.

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CHRONIC PYELONEPHRITIS

By G. A. THOMPSON, '41

Pyelonephritis is an inflammation of both the kidney parenchyma and the renal pelvis. An acute attack may clear up or it may develop into a chronic state with the signs and symptoms lasting for many years. Braasch considers the disease to be chronic when the manifestations have lasted for at least one year. Only the chronic type will be considered.

Etiology

Chronic Pyelonephritis is a bacterial infection caused by a number of different organisms either singly or in combination. It might be mentioned here that in the former type, eradication of the bacteria is easier than in a case caused by more than one organism. However, bacteria may reach the kidney without causing any demonstrable infection. Whether an infection results or not, depends mainly on the virulence and number of the bacteria reaching the kidney, and on local and general immunity of the body.

The commonest causative organism is the Colon Bacillus; others are Staphylococcus Albus, B. Proteus, Streptococcus Faecalis and S. Viridans, and more rarely Staphylococcus Aureus, Streptococcus Haemolyticus and Bacillus Typhosus. Necker gives the following figures for the frequency of the various organisms:—

B. Coli	— 74%
Staphylococcus	— 17%
Streptococcus	— 4%

The routes by which the bacteria reach the site of infection may be varied:

1. They may ascend from a lower urinary infection via the periureteral lymphatics. Some type of urinary obstruction is a common accessory factor.

2. They may reach the site via the blood stream. The bacteria may affect the renal parenchyma first and then the pelvis or the pelvis may be the primary site.

3. They may come from an extra renal infection and spread to the kidney by direct extension, or along perivascular lymphatics.

Pathology

Chronic Pyelonephritis usually involves both kidneys.

Gross—The affected kidney is usually small but it may be normal in size or even larger. As the destructive process progresses the size tends to diminish except when there is some obstruction, either functional or structural, lower down the urinary tract, in which case the kidney will be larger than normal and pyonephrosis will result. The surface of the kidney is markedly irregular due to depressed areas in which the normal tissue has been destroyed and replaced by scar tissue. There may be areas raised above the surface which consist of dilated renal tubules. This is a compensatory mechanism on the part of these tubules to increase their function in an effort to make up for those tubules destroyed by the infection. On the cut surface, there is marked narrowing of the cortex in the scarred areas. Indications of the activity of the infection are the presence of (1) abscesses, and occasionally (2) streaks of pus radiating from the region of the pelvis out towards the capsular surface. The capsule is adherent to the underlying renal tissue and when it is stripped away, bits of parenchyma are pulled away with it. The wall of the pelvis is thickened and is often reddened and covered with exudate.

Microscopic—In a *scarred area,* the following changes may be observed:

1. The glomeruli are in all stages of fibrosis with concentric thickening of the pericapsular tissue.

2. The tubules are lined by flattened atrophic epithelium and their lumina are filled with colloid material.

3. The interstitial tissue is increased and infiltrated with lymphocytes and plasma cells. If the process is active, there will be polymorphonuclear leucocytes present.

4. The vascular changes may be divided into those affecting the arteries and those affecting the arterioles.

In the *arteries*, there is an increase in the intima with narrowing of the lumen and a reduplication of the internal elastic membrane. There may be medial hypertrophy. In the *arterioles*, there is thickening of the intima. There may be an increase of collagen associated with this change.

There may be areas in which the normal tissue has disappeared and has been replaced by scar tissue.

The *non-scarred areas* may be normal or they may show patches of dilated tubules which form the raised nodular areas mentioned above.

The wall of the pelvis is thickened and fibrotic and is markedly infiltrated with lymphocytes, plasma cells and polymorphonuclear leucocytes. The surface epithelium may be covered by a fibrinous exudate or it may be ulcerated away.

Clinical Picture

This condition is not always diagnosed easily and many are found only when an autopsy is done.

In an untreated case, the disease is continuous and slowly progressive but since the advent of drugs of the sulphanilamide group, this gloomy outlook has been changed.

Although the disease is continuous and slowly progressive, the signs and symptoms vary greatly from time to time, from periods during which the patient feels relatively well, the only signs being pyuria and bacteriuria, to periods in which acute exacerbations take place with symptoms of any acute infection—chills, fever, etc.

The commonest complaints are loss of appetite, weakness, lack of energy, pallor, vague abdominal pain and a low grade fever. He will notice changes in his urine—it will be increased in amount, turbid, occasionally there may be blood and there may be pain on micturition. He will complain of day and night frequency. Oedema is not common.

As the process progresses and the damage to the kidneys becomes more extensive, such complaints as pain, fever, etc., tend to wane and signs and symptoms of hypertension and renal failure increase. Weiss and Parker estimate that 15-20% of cases of renal hypertension are caused by chronic pyelonephritis.

The commonest cause of death is uremia but this may be superceded by some accident caused by hypertension, e.g.—cerebral haemorrhage. However, Weiss and Parker in 100 cases failed to find a single death due to complications of high blood pressure.

Pyelographic examination shows varying amounts of deformity in the urinary tract. There may be no deformity (especially in the early stages) but usually there is some. The commonest findings are irregular dilatation of the ureters and calyces and constriction of the infundibuli. The pelvis may be contracted.

Urinary Picture

In this section and in the following one "Renal Function" the progressiveness of the disease must be borne in mind because as the disease continues, the amount of kidney tissue functioning becomes less and this is of course reflected in the urine and renal function.

In the earlier stages there is frequency and polyuria; the night volume is increased. However, as the disease passes on towards the end stage, the volume of urine becomes reduced. In the meantime, the daily variation in specific gravity becomes less than normal and the specific gravity of any specimen taken at random will be reduced. Both these changes will become more marked as the disease progresses.

If the infection is caused by B. coli, the urine is strongly acid, while it is alkaline if B. proteus and pyogenic cocci are the agents. The latter is due to splitting of urea by the bacteria to liberate ammonia. Albuminuria is present because of the damage to the walls of the vessels in the glomeruli; this allows the

proteins to leak out. The urine may be cloudy from the presence of pus cells and there may be frank blood from a ruptured vessel. However, such an occurrence is not common. Often the microscope must be used before pus or red cells are found.

Microscopic—The presence of pus cells is the most constant urinary finding. Bacteria will usually be found. Red blood cells are frequently present but are never as numerous as the pus cells unless a haemorrhage has taken place. All kinds of casts may at some time be seen.

Renal Function

The efficiency of the kidney becomes progressively lessened and all the functions are impaired. As has been mentioned the *volume* of urine is increased at first. This is a compensatory mechanism whereby the damaged and failing kidney tries to excrete the waste products. The *two hour test* shows a decrease in the range of specific gravity in consecutive samples. The urine becomes dilute because of increased filtration and decreased tubular re-absorption. Because of the damage to the tubules, ammonia production and possibly the conversion of dibasic phosphates to acid phosphates are both impaired and this may result in depletion of bases of the body. Finally, the elimination of waste products of protein metabolism is affected and the end result—uremia—is reached. However, it is estimated that from ½ to 2/3 of the kidney tissue must be destroyed before the N. P. N. of the blood becomes elevated.

Treatment

Medical

It is important to determine the causative organism (or organisms) since the therapeutic effect of drugs depends partly on the bacteria. For instance, Streptococcus Faecalis is almost completely resistant to sulphanilamide. The pH should also be known because mandelic acid is completely ineffective in alkaline urine; the optimum pH is between 5.0 and 5.5.

The commonest drugs used in the treatment of Chronic Pyelonephritis are those of the Sulphanilamide group and Mandelic Acid. Since the advent of drugs of the Sulphanilamide group, the use of Mandelic Acid has more or less fallen into the discard. In the use of these drugs, it is necessary to continue their administration for some time after the disappearance of symptoms and the advent of sterile urine, because it has been shown that recurrences will follow if treatment is stopped at that time.

The Sulphanilamide group is effective against B. coli, B. aerogenes and B. proteus; it is not so effective against cocci but more often than not, they will be eradicated. The efficiency varies with the amount of pathological change in the tissues; thus it gives better results in the earlier stages when there is less scarring and deformity.

The drug is given according to the "Minimal effective step—dosage regimen" of Mitchell, Greey and Lucas in which the daily amount of drug is increased to a desired quantity per day is maintained for four days after the urine becomes sterile, then reduced. The actual figures are first day—15 grs. (5 t.i.d.), second—30 grs. (10 grs. t.i.d.), third—45 grs. (15 grs. t.i.d.). The dosage can be increased still more if necessary. However, the largest dose is continued until four days after the advent of sterile urine, then it is reduced by 5 grains every other day.

This regimen is not only very effective but the toxic effects of these drugs have been markedly reduced. If after a few days on this regimen the infecting organisms (except Streptococcus Faecalis) are not erradicated, or the condition much improved, complicating conditions such as calculus, stricture, enlarged prostate, etc., should be looked for.

Mandelic acid (usually given as the ammonium salt) is only effective in an acid urine and thus it is usually given in conjunction with an acidifier (ammonium chloride). If the urine cannot be made acid, then this drug is useless. Thus it is powerless against B. proteus and some Staphylococci which are associated with

a persistently alkaline urine. This drug is non-toxic but it aggravates a diseased kidney.

Both these drugs are excreted by the kidneys and a certain concentration must be reached in the urine before they have any effect. Thus, as kidney functions fail, the efficacy of the Sulphanilamide group and Mandelic Acid decreases, i.e. the later in the disease treatment is instigated, the less are the chances of stopping its progress.

Surgical

The cause of the infection must be looked for but it must be remembered that manipulation may easily give rise to an acute exacerbation. If there is no serious condition present in the lower urinary tract, passage of ureteral catheters with lavage of the renal pelvis, using antiseptics, is a valuable procedure. If there is, however, serious complications such as hypertrophy of the prostate, stone in the bladder or stricture of the urethra, these must be dealt with to remove the obstruction, and copious fluids given to wash out the waste products which have accumulated in the blood.

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On All Idiopathic Diseases:—

"The candid reader of medical history can not but be impressed with the lack of accurate information of the causes of disease. In morbid anatomy, macroscopical and microscopical, in symptomatology and clinical description, in differential diagnosis and in knowledge of the causative connection with each other of different affections, medicine has advanced wonderfully within the last half-century. Can we say as much in behalf of etcology? Putting aside communicable diseases, a reviewal of medical literature could leave no doubt as to the answer to this question. How many diseases have been loosely attributed to "taking cold", to physical habits of life, to mental conditions, to climate; and, when it has seemed irrational to specify causative agencies, it has been necessary to say that they originated spontaneously, overlooking the fact that spontaneity is no more applicable to pathological conditions than to any other of the phenomena of nature. It is a trite truism to say that all effects have adequate causes. To say that a disease has a spontaneous origin is no less a solecism than to regard a waterfall as fortuitous, taking no account of gravitation. Instead of saying that certain diseases arise spontaneously, a more honest expression is a simple confession of ignorance."

Medicine of the Future
An Address
By Austin Flint (Senior), M.D., LL.D.
New York—D. Appleton & Co.
1886

MANIC DEPRESSIVE PSYCHOSIS

A CASE STUDY OF MANIC DELIRIUM

By CECIL E. WALKER, M.A., '41

The prime essential for the study of mental illness is a comprehensive knowledge of the individual. A further requisite is the realization that all reactions which give rise to the various manifestations of mental illness are merely exaggerations of similar reactions occurring in everyone under similar circumstances and are abnormal only in the frequency, intensity, and persistence with which they are presented.

Mental illnesses tend to develop along certain paths into more or less distinct reaction types which may be studied separately under aetiology, symptomatology, and reaction to treatment. Such reaction types are Manic-Depressive Psychosis, Schizophrenia, Paranoia, and Reactions Secondary to Organic Diseases. An attempt will be made to present a case manifesting one phase of the first-mentioned type, viz. Manic Delirium, and to show the relation of other reaction types to that case.

Case of A.B.

This young, single woman, age 24, was admitted to mental hospital on September 3, 1939, after performing an abortion upon herself leading to a septic condition. She was described as noisy, screeching, impulsive, refusing treatment, and possessing delusions that she was God and a wonderful actress. She was dehydrated and toxic.

A. led a normal, healthy, happy childhood in a large French-Canadian family of good economic status. The father, a builder and contractor, had been in poor physical health for several years. The mother enjoyed good physical health although emotionally unstable, subject to persecutory ideas, being agitated and depressed for about a year during the menopause with later complete recovery. There is no other history of mental illness in the family.

After completing her entrance at age fourteen, A. became a salesgirl, doing very well and enjoying her work in which she continued up to the time of her misfortunes. She was fond of people, especially children. Last year, after quarrelling with the young man she was going to marry, she became melancholy at times but was usually cheerful, witty, and emotionally excitable. Swimming, cycling, and hiking were favourite pastimes. Sexual history was normal until the present situation. She was not troubled by religion, seldom drank, but got drunk easily.

In May, 1939, a young male acquaintance, deceiving her by promises of getting her a better job, coaxed her to attend a party with him. He gave her a potent drink. At the end of the evening he told her that everything would be all right and that he had taken the necessary precautions. Nevertheless she became pregnant and was deserted by the young man. Becoming desperate, she aborted herself with a toothbrush but kept the facts from her parents. Profuse bleeding necessitated her entrance to a general hospital where she progressed rapidly, showing no mental unbalance until her old boy friend came to visit her, when shame and guilt were brought home to her. Soon the picture of delirium developed.

A. became irrational, demanding, impulsive, striking the nurse, refusing all food, necessitating her removal to a mental institution. Tube-feeding was a regular necessity, the patient gradually losing weight. There were rational intervals in which she smiled, asked names and remembered for a short time. Delusions of being God and a great actress appeared. Hallucinations were not in evidence. The delirious state at the time led to a diagnosis of Toxic Psychosis.

Later, however, everyone agreed that there was no longer a septic condition but a disturbance of physiological mechanisms accounted for the wide swings of temperature rising to 106 degrees at times. There were no signs of toxaemia, no fall in blood pressure, no increased white count, and no positive findings on gynecological examination. However, the delirious state has persisted unabated to the present. Menstrual periods have not reappeared since abortion.

Still later, a marked pressure of speech with very obscene language and flight of ideas appeared. The temperature gradually decreased but excitement and activity increased. However, in lucid intervals it is evident that the patient is in touch with her surroundings and is not completely confused. Treatment with sedatives, continuous water baths, and tube feedings have continued intermittently to date.

Before describing the essential picture of Manic Delirium let us discuss the relationship which two conditions viz. Schizophrenia and Toxic Psychosis may have to this case.

Schizophrenia Discussed

Schizophrenia, in its typical form, consists in a slow, steady deterioration of the entire personality, usually showing itself at the period of adolescence. It involves principally the affective life, and expresses itself in apathy, in disorders of conduct and of thought, and in an increasing withdrawal of interest from the environment. There is usually an early history of continued inability to adjust to social demands with a consequent withdrawal into a world of phantasy. The definite psychotic reaction is often precipitated by conditions of stress such as toxic-infective illness, pregnancy, economic worry, love affairs, etc.

However, in this patient we find no evidence of early maladjustment and "introversion". The sudden onset of symptoms in an emotionally high strung woman, the absence of apathy, and no sign of "splitting" of the personality (which is the meaning of the term Schizophrenia) all point away from a Schizoid state. The age of the patient and the traumatic factors of sexual mishap and resultant abortion nevertheless necessitate an open mind to the possibility of this syndrome appearing in a later picture.

Toxic Psychosis Discussed

This group includes mental disorders produced by infections of various types, septic abortion being the one under discussion here. They are often characterized by delirium such as depicted in the history. Wild excitement, illusions, and hallucinations of sight and hearing, and fear are common symptoms. If the psychosis persists beyond the febrile stage, apathy, loss of memory, or possibly depression are likely to result. It is in this febrile state that such syndromes as Schizophrenia and Manic Depressive Psychosis are unmasked. It is believed the latter change has occurred in this patient.

Although the early picture in this case would seem to point towards toxic delirium, the persistance of delirium long after physical disease had abated, the characteristic unfolding of the manic state to be described, and the exaggeration of the mental responses far beyond the bounds of the relatively slight organic involvement, all point away from a toxic state as the whole explanation. The toxic state, however, may be looked to as a chief precipitant for the picture to follow.

Manic Delirium Discussed

In this discussion we shall attempt to present an organized picture of this syndrome.

General

The term Manic-Depressive Psychosis was introduced by Kraepelin to characterize disorders of affect (i.e. "emotion" or "feeling") consisting either of elation or of depression. Thus, two general phases are recognized—Manic and Depressive. Manic Delirium is a special form of the first phase.

Aetiology

1. Heredity—This is important and involves the mental stability of the predecessors.

2. Constitution—There is usually a mental make-up in which the affect swings from elation to depression in people with frank, open personalities. They are either bright, talkative, optimistic, aggressive people who make light of the ordinary affairs of life, or else they take a gloomy outlook; or there may be an alteration or combination between the two states.

3. Physique—No convincing evidence of physical deviation from normal has been shown.

4. Precipitating factors—e.g. business worry, sexual trauma, toxic conditions.

Symptomatology

1. Mental signs and symptoms

(a) Manic States in general

1. Elated though unstable mood.
2. Flight of ideas as shown by continuous but fragmentary stream of talk.

3. Increased activity (psychic and motor). Irritability, suspicion, clouding of the consciousness, with delusions, hallucinations, disorientation, and lack of insight may also appear ,but are usually transitory.

(*b*) *Manic delirium*

Symptoms of (a) very marked with added:

1. Total disorientation for time, place, and person.
2. Incoherent conversation.
3. Neglect of personal appearance.
4. Total lack of insight.

2. Physical signs and symptoms

No characteristic symptoms. But we find:

1. Disorders of sleep.
2. Emaciation due to lack of food.
3. Cessation of menstrual periods and absence throughout the illness.

Diagnosis

A history of acute onset in a person formerly considered quite healthy though emotionally unstable.

Progress of Manic Delirium

This is usually favourable. Attacks may last three to six months or more. Much depends on the previous personality. Cases starting after age 40 carry a graver prognosis. Those not clearing up tend to show gradual deterioration with well-preserved intellect.

Treatment of Manic Delirium

Specific Treatment—none.

Symptomatic Treatment:

1. Careful observation in a mental institution.

2. Confinement to bed with reduction of exciting influences.

3. Light nutritious diet, tube-feeding usually necessary with periodic washing out of the stomach.

4. Continuous water bath provides a means of partial restraint, the heat having a sedative effect.

5. Hypnotics for extreme restlessness or sleeplessness: Paraldehyde, drachms ii to iii is the drug of choice, or sodium barbitol gr. x-xv; potassium bromide gr. xx by itself or bromide and chloral gr. x-xv.

6. In emergencies, a hypodermic of H.M.A. may be given (an H.M.A.= hyoscine gr. 1/100, morphine gr. 1/6, atropine gr. 1/200).

7. Thorough study of the underlying causes of the case.

Final Note

The reader has been presented with a discussion of three mental reaction syndromes, the last one being treated in detail. He may now gain a clearer conception of the mental state of the patient, the causative factors, the symptoms, the prognosis, and the best course of treatment to be undertaken. It is felt by the writer that the patient's condition now parallels very closely the description outlined for the Manic Delirium. The treatment employed at present is essentially that sketched in the last section.

THE TREATMENT OF EARLY SYPHILIS

By R. H. FERGUSON, '42

Early syphilis includes primary cases and secondary cases of less than two years duration. This subject has special significance at the present time, in that during war, syphilitic infection tends to become more virulent and progressive.

In untreated syphilis the primary chancre appears about three weeks after infection has occurred, and about one week later the blood Wassermann test become positive. After six to eight weeks, when the chancre begins to heal, secondary lesions appear in the skin and mucous membranes and elsewhere. These persist for a variable term, ranging from a few days to several months, and disappear spontaneously.

Antisyphilitic Drugs

Arsphenamine (606, Salvarsan)—was discovered in 1910 by Ehrlich and Hatta. It is the arsenical of greatest therapeutic value. Owing to the necessity of carefully neutralizing it with sodium hydroxide and administering it intravenously in well diluted form by the gravity method, it has been used less commonly in recent years. It is given in doses of 0.3 gm. to 0.6 gm. once weekly.

Neoarsphenamine (914)—also introduced by Ehrlich and Hatta, is the most widely used arsenical. It has the advantage of injection into the blood stream without the need of neutralization. Each dose is dissolved in distilled water and given intravenously by the syringe method. The dose is 0.3 gm. to 0.9 gm. every five or seven days.

Arsphenamine and Neoarsphenamine are yellow powders which are readily oxidized on exposure to air, forming highly toxic products, and are therefore sealed in glass ampules.

Sulpharsphenamine is closely related to neoarsphenamine but has the advantage of intramuscular injection. Although therapeutic results are good, it is more prone to produce undesirable reactions than neoarsphenamine. Its use has become restricted to cases where intravenous treatment is not practicable, as in treatment of infants or of persons having small or inaccessible veins. It is given in doses of 0.12 gm. to 0.48 gm. at weekly intervals.

Arsenoxide as represented by Mapharsen, is a new drug, giving some promise of being of real value. It is given intravenously in weekly doses of 0.03 gm. to 0.04 gm. dissolved in distilled water.

Bismarsen contains an arsphenamine base in combination with bismuth. It is given by intramuscular injection in doses up to 0.2 gm. once or twice weekly.

Stovarsol is a pentavalent arsenical compound that is given by mouth. It is used in the treatment of young infants.

Bismuth was introduced in 1921 for the treatment of syphilis as an adjunct to the arsphenamines in place of mercury. It has a definite spirillicidal effect, although less than the arsphenamines, and also encourages resistance building, which is very important in the cure of syphilis. It is usually given intramuscularly. Bismuth oxychloride and bismuth salicylate in suspension are commonly used, although several other preparations are satisfactory. Bismuth does not act as rapidly as the arsphenamines, but since it is absorbed slowly from the deposits in the muscles, its action is more prolonged. Bismuth is eliminated slowly, in urine and feces, and opportunity should be given at intervals to permit elimination of that which has accumulated in the tissues.

Mercury has been almost entirely replaced by bismuth in the treatment of early syphilis. It is not a spirillicide and treatment complications are more common than with bismuth.

Iodides have no specific action. They promote the absorption of gummatous or

fibrous areas and so are useful in treatment of late manifestations of the disease.

Methods of Treatment

A complete medical examination should precede any syphilitic treatment, and the treatment should be adjusted to suit each individual case. Neoarsphenamine intravenously and bismuth intramuscularly are recognized as the drugs of choice.

For purposes of treatment, patients are divided into three classes.

Class A — Sero-negative Primary Cases. Primary lesion present, dark field positive, Wassermann negative.

Class B — Sero-positive Primary Cases. Primary lesion present, dark field positive, Wassermann positive.

Class C—Secondary Cases. Secondary lesions present or completely healed, Wassermann positive.

Two satisfactory courses will be briefly outlined.

Continuous Method

Class A—A series of thirty to forty weekly treatments is given, the first fifteen weeks with neoarsphenamine and bismuth together, then four weeks of bismuth alone, followed by four weeks of neoarsphenamine alone, and the remainder with neoarsphenamine and bismuth. A rest period of four weeks is then permitted, following which serological tests on blood and spinal fluid are performed. An observation period of two years is now necessary, during which the blood is tested frequently.

Class B and *Class C*—Treatment is begun as in Class A, and following the rest period it must be continued, even though the serological tests are negative. A series of ten weekly treatments with neoarsphenamine and bismuth is given, followed by four weeks rest period and serological tests. This series is repeated and if, at the end of this time, the blood is negative, the two year observation period is commenced.

Intermittent Method

Class A—A course of twelve weekly treatments is given, the first seven with neoarsphenamine and bismuth, the next two with potassium iodide, and the remainder with neoarsphenamine and bismuth. This is repeated twice, with rest periods of four weeks between courses. Blood tests are made one week after the last dose of each course.

Class B and *Class C*—Three courses are given following the first negative blood test, with rest periods of four weeks between courses.

If at the completion of either of the above schedules of treatment the blood is positive, the patient is classified as a tertiary syphilitic case, and further treatment is indicated.

Many other plans for treatment have been tried, using various combinations of arsenicals, bismuth, mercury and iodides. Some experts believe that treatment should be absolutely continuous without rest periods of any kind, and that to accomplish this, courses of an arsenical must be alternated with courses of a heavy metal.

Complications of Treatment

Arsenicals

Pain at site of injection—If the needle slips from the lumen of the vein and some of the solution is injected subcutaneously, the patient will immediately experience a severe burning pain, which may be relieved by subcutaneous injection of 5% sodium thiosulphate solution into the area of extravasation.

Gastro-intestinal reactions — Nausea and vomiting either during or shortly after the injection are the commonest symptoms of toxicity. These may be avoided by giving treatment several hours after a meal and by correction of constipation.

Nitritoid crisis—This is a marked reaction associated with fall in blood pressure, and which may result in unconsciousness. Injection of adrenaline is the best treatment.

Liver damage—Persistent loss of appetite together with nausea or vomiting may denote the onset of serious liver damage. Complications may vary from

a transient jaundice to acute yellow atrophy.

Dermatitis—Itching with an eczematous-like eruption may be the forerunner of a severe dermatitis, and is the signal for change in treatment.

Herxheimer reaction—This is an intensification of inflammation in syphilitic lesions.

Fever and chills frequently occur.

Bismuth or Mercury

Immediate reactions are usually due to errors in technique of intramuscular injection. The piston of the syringe should be drawn out to see if the needle has entered a blood vessel. If an injection embolus is carried to the brain or lung, serious results will follow. A local abscess may form at the site of injection due to faulty aseptic technique. Stomatitis, as evidenced by a blue line at the gum margin, may be largely prevented by careful dental hygiene. The first evidence of kidney damage caused by mercury or bismuth is polyuria.

Results of Treatment

The great majority (80%) of early cases may, if properly treated, reach a stage where further treatment is not necessary, and where life-long observation will not reveal recurrence or other evidence of the presence of the disease.

The organisms usually disappear in twelve to twenty-four hours from the primary lesion, which generally heals in three days to a week after the first administration of an arsphenamine. Secondary lesions heal more slowly, requiring at times a week or two, but pigmentary changes may last much longer. At this time, when all lesions have healed and he feels perfectly well, the patient may think that a permanent cure has been established, and will neglect further treatment unless followed up carefully. A positive blood reaction in primary or secondary syphilis may become negative after three or four weekly injections of an arsphenamine, but unless treatment is continued, the reaction seldom remains negative.

Free treatment is in effect in every province in Canada. The object of this is two-fold; cure of existing cases, and prevention of the occurrence of new cases. It is believed that the more promptly cases are brought under treatment, the fewer fresh cases will arise. It is important that the disease should be diagnosed and treated as early as possible, thus preventing occurrence of late manifestations. The cost of treating early syphilis is only a small fraction of the expense involved in hospitalization of patients suffering from general paresis. The value of free treatment is indicated by a very definite reduction in the incidence of syphilis.

In 1905, Osler stated that syphilis is "one of the most amenable of all diseases to treatment". If all the available knowledge were applied, the disease could be almost completely stamped out in one generation.

The author wishes to express his sincere appreciation to Dr. E. J. Trow for his valuable assistance and helpful criticism in the preparation of this paper.

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Abstracts

CRAWFORD B. SHIER, Editor

Contributors A. M. Crossland, P. T. Greenberg, F. L. Lawson, R. L. MacMillan, G. R. Walker, J. G. Watt

THE TREATMENT OF CHRONIC EMPYEMA BY CONTINUOUS HIGH VACUUM SUCTION

J. V. H. NEVILLE,
Surg. Gync. and Obst. 69:241-246 Aug. 1939

The method employed for the collapse of chronic empyema cavities, described in this paper, avoids the extensive thoracoplastic operations. Obliteration of the cavity, the objective of surgery, is attained in this method by constant suction applied to the interior of the cavity. In the surgical procedure the approximation of the two walls of the cavity and its consequent obliteration is made at the expense of the outer wall. In the method described the high vacuum created within the cavity allows for the utilization of atmospheric pressure acting both on the outer wall and, owing to the pressure within the respiratory passages, inner wall. Since the ribs oppose a greater resistance to pressure, the inner wall is more readily displaced.

Suction is obtained by means of an electrically operated vacu-aspirator, an instrument which delivers a high degree of vacuum. The instrument is small, portable, and noiseless. It can be operated continuously for months with very little attention. This method of therapy deserves a trial before radical, hazardous surgery is resorted to.

A. M. J. C.

INADEQUACY OF INJECTION TREATMENT OF HERNIAE

ROBERT SLATER
New England J. Med. 221:695: Dec. 1939

The end results of two years follow up of twenty patients who have received a complete course of injection treatment for inguinal hernia (ten direct and ten indirect) is reported.

All were considered cured at the time of discharge (one to two years after the first injection). In four years the apparent cures were reduced to two!

The injection treatment is likely to fail because—(1) the plane of injection is often not determinable, (2) unless the inner lining of the sac is obliterated peritoneal pressure will force it through the fibrous tissue sooner or later, (3) injection into the sac itself is inadvisable owing to possible intraperitoneal reaction and shock.

A. M. J. C.

ROULEAUX FORMATION IN FRESH UNMODIFIED BLOOD AS A DIAGNOSTIC TEST FOR HEMOLYTIC ANEMIA

WILLIAM DAMASHEK
New England J. Med. 221:1009

Recent studies indicate that spherocytosis is dependant on the action of various hemolytic agents on mature erythrocytes, and not on abnormal formation of red cells in the marrow. Spherocytosis is not pathognomonic for congenital hemolytic jaundice as it is found in various hemolytic syndromes.

Normally, in rouleaux formation the cells can be seen as uniform in thickness, and form long, more or less straight chains, like a pile of coins.

Where there is spherocytosis the cells cannot form proper rouleaux because of their shape—they form chains that are short, bent or often bizzaire in appearance, and in which the variation in the thickness of the red cells is apparent. The more severe the spherocytosis, the more apparent this picture is.

P. T. G.

SOME OBSERVATIONS ON HEADACHE

D. W. C. NORTHFIELD

Brain, 1938, 61:133-162

Experiments were performed in order to determine the sensitiveness or otherwise of the cranial contents, and to elucidate the factors which provoke or relieve headache. Stimulation of the dura mater by various means elicited pain sensations. The tentorium and around the middle meningeal vessels were most sensitive, the dura lining the vault being least sensitive. Stimulation of the internal carotid artery also gave rise to pain. The pia mater, the brain tissue and the ventricular walls were apparently insensitive.

In some cases raising the intracranial pressure provoked or aggravated headache, but in many headache was produced by suddenly reducing the intracranial pressure. It was shown experimentally that the sensitive structure is not within the ventricle, but that the mechanism must be related to the covering membranes with their vessels and sinuses. It was concluded that the experimental headache was induced by the sudden variation in the intracranial pressure not by the steady pressure itself.

Histamine, a vasodilator, on injection causes a fall in systemic blood pressure and cerebral vasodilatation with resultant increase in intracranial pressure. During recovery from this condition, i.e., during the fall of intracranial pressure, headache results. Experiments demonstrated that this histamine headache depends primarily on an alteration in calibre of the cerebral blood vessels, and not on the meningeal vessels of the dura mater. The evidence indicates that the afferent pathway for the pain is probably along the trigeminal and upper cervical nerves.

It was concluded that the dura mater is not the sensitive structure which is responsible for the characteristic headaches of intracranial tumour. The view was advanced that such headaches are due to some disturbance of the tension of the walls of the cerebral vessels, by change in intravascular pressure, vascular tone, or extravascular (intracanial) pressure. Finally, it was pointed out that the common headache due to intracranial tumour, to an epileptiform convulsion, or to an intracranial aneurysm, satisfies remarkably fully the criteria of visceral pain, the manifestation in consciousness of a protective mechanism.

F. L. L.

GASTRIC PERISTALSIS

STUART GORDON AND
ARTHUR C. SINGLETON, *Toronto*

Surgery—November, 1939, pp. 697-703

It is commonly accepted that gastric peristalsis is a function of the entire stomach wall. Other views have been stated, chiefly that of Cole and Forsell, that the muscularis mucosa alone was capable of producing peristalsis.

The serosa and muscularis propria (two layers) are closely connected, as are the mucosa and muscularis mucosa. Between these two groups lies the lax submucosa allowing free movement between the two inner and three outer coats.

Lead beads and, later, small lighter steel beads, were sewn to the serosa of a dog's stomach along the greater and lesser curvatures. After recovery from the operation, a barium meal was given and the position of the beads examined fluoroscopically and by plates. It was found that moderate peristalsis could occur without disturbing the beads and that they were disturbed only in marked peristalsis.

The distal half of the stomach was denuded of serosa and muscularis propia. It was found that very little peristalsis occurred in this denuded area.

Conclusion:—

1. There are two types of gastric peristalsis.
2. The first type involves all the coats of the stomach wall, is probably propulsive, and is quite widespread.
3. The second type involves only the mucosa and the muscularis mucosa, but requires that the serosa and the muscularis propria are present.
4. Accompanying this second type of peristalsis, tonic changes occur in the muscularis propria.

G. R. W.

EVALUATION OF ENDOGENOUS INSULIN SECRETION

M. M. MILLER, O. P. ALLEN
Arch. Int. Med., October 1939

This method depends on Hartmann's formula for the total amount of insulin required to reduce the blood sugar to a given level. Hartmann assumes that soft tissues make up two-thirds of the body weight: also that the sugar content of these tissues is the same as the blood sugar level: and finally that 1 unit of insulin oxidizes 2 grams of glucose. Thus the insulin needed to lower the blood sugar from *A* to *B*.

$$= \frac{\text{A-B grams} \times 2/3 \text{ body weight grams}}{100 \times 2}$$

units where *A* and *B* are blood sugar figures in milligrams per 100 cc. of blood.

To calculate the endogenous insulin all factors involved in carbohydrate metabolism are lumped together as if the pancreas alone is involved. The total amount of insulin required is estimated by the above method and then a smaller dose of insulin is injected. The fall in blood sugar is measured and from this the amount of glucose oxidized can be calculated. Dividing this quantity by 2 gives the total number of units of insulin used. The difference between this and the amount of insulin injected represents the endogenous insulin.

R. L. M.

EFFECT OF DISEASE OF THE LIVER AND BILIARY TRACT UPON THE PHOSPHATASE ACTIVITY OF THE SERUM

ALEXANDER B. GUTMAN,
KENNETH B. OLSON
ETHEL BENEDICT GUTMAN
CHARLES A. FLOOD
Journal of Clinical Investigation

An analysis has been made of 350 cases of disease involving the liver and biliary tract, in whom the diagnosis could be proven, with regard to the influence these affections had upon the level of serum phosphatase activity in the adult.

"Alkaline" serum phosphatase originates mainly in bone-producing cells, is carried in the blood to the liver, and excreted in the bile. Elevations of serum phosphatase activity occur in: (1) diseases of the skelatal system, and (2) diseases of the liver and biliary system, with obvious or presumptive impingement on the excretory tract. The first results in increased formation, the second in retention, of the enzyme. An alternative theory is that disease of the liver results in increased activity and formation of the enzyme, by irritation of the skeletal system by some activating substance retained in the blood.

The normal serum phosphatase level in adults is 1-4 Bodansky units per 100 cc. serum.

Conclusions:

(1) A value of less than 10 Bodanski units of serum phosphatase activity makes unlikely a diagnosis of obstruction of the extra hepatic biliary tract.

(2) A level higher than this is supportive, but not positive, evidence of extra hepatic biliary obstruction. Other causes of such a level might be liver abscess, liver secondaries, and a few cases of catarrhal jaundice, or jaundice following the injestion of hepato-toxins, e.g., arsenic, or disease of the skeletal system.

(3) It is suggested that the test be used to provide positive evidence of obstruction, in conjunction with the liver function tests, which provide only negative evidence of obstruction, and the dye tests, which are inapplicable in jaundiced patients.

G. E. W.

THROMBOPHLEBITIS

The Role of Vasospasm in the Production of the Clinical Manifestations

ALTON OCHSNER, M.D. AND
MICHAEL DEBAKEY, M.D., *New Orleans*
J.A.M.A., Jan. 13, 1940

In their opinion the concept mechanical blockage of the venous and lymphatic systems is of primary significance in the production of the clinical manifestations of thrombophlebitis is inadequate.

Based on recent clinical and experimental investigations they believe that many of the symptoms and signs are due to vasospasm, which was first suggested by Leriche, and that the vasoconstricting impulses arise in the thrombophlebitic segment. As a result of the vasospasm there is increased filtration pressure, relative anoxia of the capillary endothelium and diminution in the flow of lymph, all of which increase the amount of perivascular fluid.

Fifteen patients with seventeen thrombophlebitic processes have been treated with procaine hydrochloride block of the sympathetics. There was prompt and permanent relief of pain in all instances. In half the cases the temperature returned to normal in forty-eight hours or less and the other half were normal within a week. In more than half the cases the oedema completely subsided in eight days and the remainder within twelve days. Sixty per cent of the patients were discharged from the hospital as cured within eight days of the institution of therapy. The earlier this treatment is begun the better is the result.

The number of injections in each case varied from one to six. When the lower extremity was involved the lumbar sympathetics were blocked with one-per cent procaine hydrochloride, the needle being run through the dorsal musculature two and one-half fingers' breadths beyond the process of the second lumbar vertebra. The Stellate ganglion is blocked for thrombophlebitis involving the upper limb.

J. G. W.

Book Review

FUNCTIONAL DISORDERS OF THE FOOT

By F. P. DICSON AND R. L. DIVELEY
Publishers: Lippincott, Philadelphia

Two important monographs upon the foot have recently been published. The first was that of Morton ("The Human Foot"—Columbia University Press), a scholarly scientific enquiry into the structure and physiology of the foot with certain important excursions into pathology. This has done much to clarify certain obscure foot problems. The second is that of Dickson and Diveley, reviewed here.

"Functional Disorders of the Foot" is the most important monograph on the clinical aspect of foot problems which has been published in recent years. It is intended for the student, the practitioner and the orthopaedic surgeon and it fills an important need. The elaborate discussion on the evolutionary development of the human foot which so often precedes any such monograph, and completely confuses all but anthropologists, is here reduced to a brief chapter. The remaining chapters discuss the anatomy and physiology of the foot followed by chapters discussing the various kinds of foot disabilities. Perhaps the most important point it drives home is this: flat foot or foot strain can be due to many causes though the result is similar in each case. The exact cause of each case of foot strain must be sought if adequate treatment is to be given. There is careful description of all the important causes of foot disability. Treatment is adequately covered and emphasis is laid on simple forms of treatment.

A most important monograph likely to be of great value to every practising physician.

R. I. HARRIS, M.D.

Feature Section

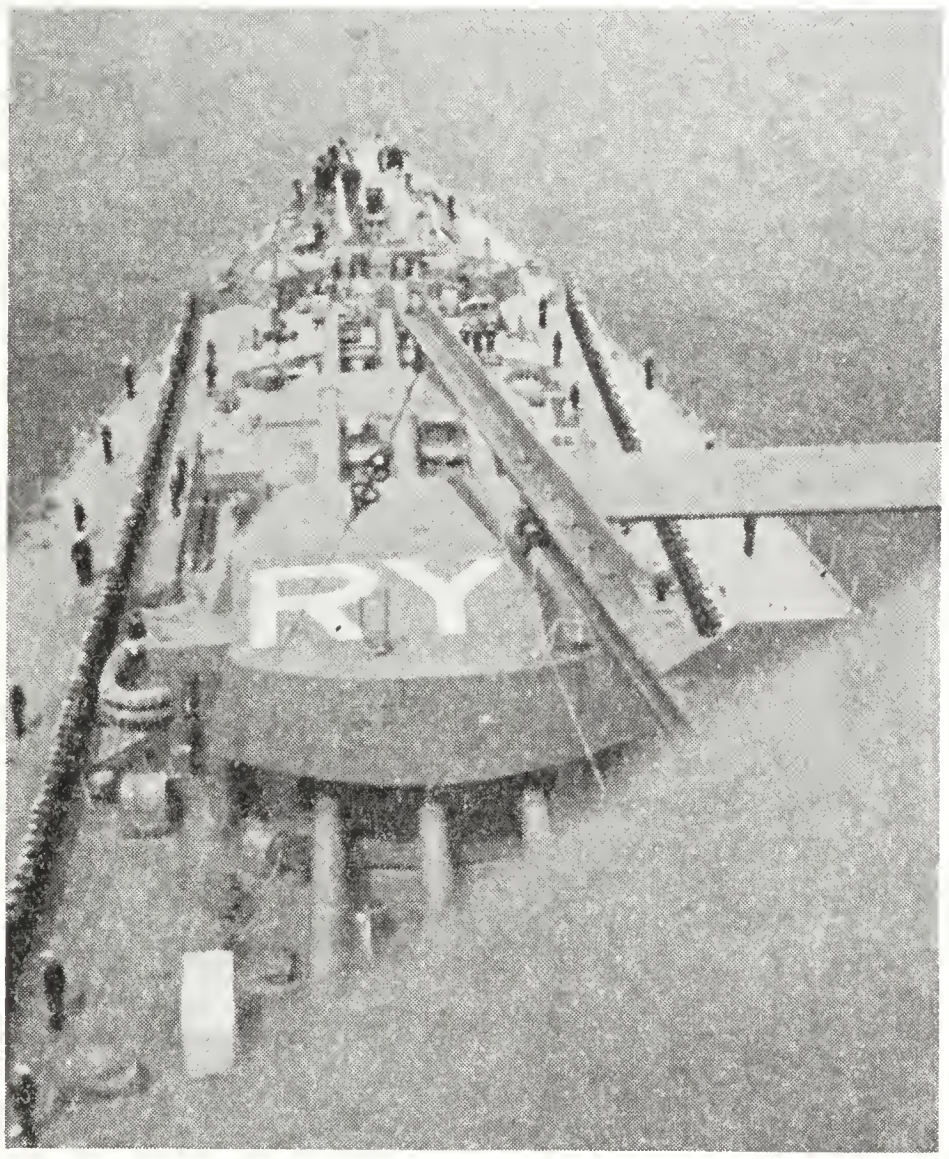
F. L. LAWSON, Editor

TEN DAYS ABOARD A BRITISH BATTLESHIP

By H. A. SIMS, B.A., '40

Whenever your Editor and I have mentioned our cruise in H. M. S. Rodney, the first question which has always been asked is how we managed to arrange the trip. Therefore it seems appropriate to commence this present account of our travels with an explanation.

school boys to spend two days with the Navy for the Coronation Naval Review. He managed to arrange a similar trip for some of his charges in 1938. The Admiralty gave him berths for thirty-five boys for a cruise of ten days to two weeks' duration, to start early in July.



A Naval Salute to Skagen, Denmark.

Ken Greer and I were in England in the early summer of 1938 after a crossing in a cattle boat similar to that described by Al Lane (*U. of T. Med. J., Feb., 1939*). In London we met Major Ney, the guiding spirit of the Overseas Education League. The year before, he had arranged for a number of Canadian

Some of the boys had not yet arrived in England, and the Major suggested that we take the trip. Our only expense was to be a nominal amount for messing and we were to be associated with the Midshipmen and the younger Sub-Lieutenants, whose mess is the Gunroom. This is so called because in the old sailing

ships, it was in part of one of the gun decks.

The *Rodney*, along with about six other big ships, including the unfortunate *Royal Oak*, was at Greenock in the Clyde for the Glasgow Exhibition and we were to join her there early in the morning of July 4th just before she sailed. We left Glasgow at five a.m. on the 4th in a train full of returning seamen bound for Greenock. We found a picket boat from the *Rodney* waiting for us at the pier at eight and our naval adventure was finally begun. We were taken to the after ladder of the ship and started up. An officer above shouted to us to run up, which we painfully attempted to do. At the top we found a guard of seamen drawn up, with the officer saluting. As we were without hats we could neither acknowledge the salute nor honour the quarterdeck itself. This latter duty dates from the time when the quarterdeck bore a crucifix. The Sub-Lieutenant seemed a little nonplussed to find that the expected schoolboys were at least as old as himself and he requested a midshipman to show "these" to the Gunroom.

The Gunroom is about thirty feet square and acts as dining-room, study, common-room and home for about thirty midshipmen and three Sub-Lieutenants. The *Rodney's* Gunroom is the largest in the fleet in proportion to the ship's size. We were very well received by the denizens of the Gunroom and made to feel quite at home—but for several hours we were not allowed on deck, until word was received from the Captain as to what restrictions were to be placed on our movements. We had to be satisfied with glimpses through the scuttles (portholes or plain windows, to us) of the fleet sailing and leaving us. When finally we were allow on deck in the afternoon, the ship was approaching Bangor, a little seaside town down the Lough from Belfast.

We stayed at Bangor for four days, on a sort of goodwill visit. Some of the officers played cricket matches ashore, the Captain entertained the civic officials, and a large reception was held on board for all the elite of the district. The Ulster girls were conceded by all to be very attractive, and fine dancers as well, but Ken, departing entirely from type, spent the afternoon with *one* older woman. However, he simply made up for lost time later in Denmark. We made several excursions ashore, including one brief visit to Belfast, and an all-day trip through Belfast to Port Rush on the north coast, where we visited the Giant's Causeway. Here Ken took it into his head to scramble up a tremendous cliff, which looked, however, more a test of endurance than of mountaineering skill. Afterwards, we found the natives looking on him in a sort of awe. They explained that it had never been scaled before, and the last man to attempt it, about two years before, had been killed! We were interested to see at Port Rush our sister ship, *H.M.S. Nelson*, which was making a similar visit there.

We got back to Bangor to find a miserable rain, a howling wind and a high sea breaking on shore. The thirty-foot picket boat which we expected to take us back to our ship, was at the dock, but had already failed to make the grade and was waiting the night, so we had to go in the launch. This is a great overgrown eleven-ton dory, completely open and fairly heavily powered. It is used for taking the men ashore and will hold about two hundred. There were about fifty of us that night and we hung on for dear life as the boat was picked up by the waves and dropped again like a toy. Every time it came down we expected the bow to disappear for good, but instead of cutting in, it fell flat on the waves with a smack which sent clouds of spray over us. Even when we reached the ship's side our troubles were far from over, because the boat was bobbing a good five feet above and below the boarding platform. All thought of precedence Twenty-five—med journal—3933 yDo .. in leaving boats was forgotten, and whoever was nearby when the platform passed us, made a jump for it, and was caught by two seamen who clung to the step. The only casualty was one seaman's cap, knocked off as he jumped.

The following afternoon we bade Ire-

land goodbye. With a battleship this is not taken lightly. Whenever the ship leaves port, all hands are mustered on the fore-deck, and the band plays while the anchor is weighed and she steams slowly out. During such ceremonies, we civilians were not to be in sight, but we found that the very top deck of the main tower gave a splendid view, without making us very obvious. We were at sea from Friday afternoon till Monday morning. We went up among the Orkneys and Hebrides, through the Pentland Firth, unfortunately at night, across the North Sea, skirting Norway, to Skagen at the northern tip of Denmark.

While at sea, we had a chance to look around a little and get to know the ship. *Rodney* displaces 33,000 tons. It was originally planned for 44,000 tons, but along with *Nelson*, is known as the Cherry-tree Class, because it was cut down by the Washington Treaty! This accounts for its peculiar shape with the big guns all grouped for'ard, to save in the weighty sixteen-inch armour. The *Nelson* being flag-ship is more crowded with offices so the main hospital for the home fleet, and the chief surgeon, are to be found in the *Rodney*. She has a good operating room, X-ray equipment, and a fair sized sick-bay. The Surgeon-Commander apparently learned his anatomy the hard way, because he objected to Grant's *Method of Anatomy*, that it read like a story-book! In addition to the Commander there were two Surgeon Lieutenant-Commanders, and a Dental Lieutenant-Commander. They had the care of a ship's company of eighteen hundred men. As well as the regular medical other ranks, the mess stewards help in the dressing stations during action. These stations must of necessity be distributed over the seven hundred-odd feet of the ship at various levels because in action the watertight bulkheads and hatchways are closed to localize any damage. One surprising feature is the presence in the ship of an elaborate liquid-air plant to supply the fleet with oxygen for emergency welding. We were interested to learn that the Royal Marine detachment is quartered between the officers and the seamen, traditionally to quell mutiny.

Like the midshipmen, we were given hammocks in which to sleep. These introduced unexpected technical difficulties. They are all hung in one big room, from iron bars projecting from the ceiling. The correct way of mounting is to swing by both hands from the bar at one end and ease yourself in. The trick, and not an easy one, is to avoid tipping the beastly thing and falling the five or six feet to the steel deck below. Once safely in, the next trick is to learn how to breathe against the pressure of the sides, which squeeze in on you, because, unlike a garden hammock, it is hung from a single rope at each end with no wooden spreader to maintain the width. Finally, you must try to keep your covers from falling off your few inches of bed. This I never did learn and the Royal Marine sentry spent the night picking up my blankets. Then we were awakened at an unearthly hour every morning by having the deck scrubbed beneath us, with much chatter and rattling of pails. The other noises which challenged our sleep were the constant "machinery murmur" of the ventilators and the announcement system which relayed orders all over the ship. This latter was about as musical as the one in the Children's Hospital, but the bos'n was not as good at holding his final notes.

There seemed to be many things to occupy our days. We spent hours just talking to the fellows in the Gunroom, who were a very nice friendly crowd. They seemed very earnest and hard-working but were certainly not above a bit of fun on occasion. While we were at sea they organized deck-hockey among the officers and petty officers. This is an informal kind of shinney played with great heavy crooked canes and a spliced rope ring or grommet. One man was kept continuously occupied making new ones to replace those lost in the sea. Strangely enough, though, we did not lose any men that way.

We were allowed to ramble almost without restrictions about the ship and the only limitations even on photography

were close-ups of special equipment and the use of telephoto-lenses. We spent several watches on the bridge where we learned a little of the mysteries of moving the great fortress about. The wheel-house is a separate compartment below the bridge, lightly armoured and without windows. The steering is entirely by compass, the course being automatically indicated from above. The helmsman let Ken try his hand at steering one day. The limit of swing allowed is one degree on either side of the course, but Ken found ten degrees more his speed, so when the officer of the watch noticed his precious 33,000-ton charge weaving in twenty-degree arcs across the North Sea, your Editor's career as a navigator was quickly terminated. The Captain remains aloof from the general routine of the ship. The second in command, the Commander, attends to these matters. Our first meeting with the latter was when he reprimanded us for strolling the deck with our hands in our pockets, and for leaning on the rail, both breaches of naval etiquette.

Approaching Skagen was an interesting procedure. It is the boast of the Royal Navy that they never use local pilots, but can feel their own way into any port. The bottom at Skagen is very long and shelving and we finally came to rest about a mile from shore with, even there, only a couple of fathoms to spare at low tide and shallower water still on either side. To guide him, the Navigating Officer made use of lead-line soundings, an acoustic sounding device, the ordinary crossed bearings on objects ashore and even of the big twenty-foot range-finder for the guns. This last we felt was taking unfair advantage of merchant ships not so equipped.

At Skagen our life with the Navy came to an end. The ship was staying there for nine days and our time was short, so we left her after four days and travelled back through Denmark ourselves. The Captain entertained us at a very lavish lunch in his cabin on the day we left and we were sent ashore in a special picket-boat. The final surprise was when two greasy "stokers" or engineers climbed from the tiny hatch of the boat to bid us goodbye! They turned out to be a pair of our Gunroom friends spending the day studying boats' engines.

We left the ship with very pleasant memories of the men of the Senior Service whom we had met and with a very real respect for the efficiency and formidability of that Service.

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Medical Society Notes

By PAGE STATTEN, Secy.-Treas.

At the time of writing, the Meds-at-home has passed into our happy memories. We join with those students who were able to attend in extending our congratulations to Bill Ortved and his committee on the real success of the party. After attending the dances of the past few years which were publicized with such names as Bunny Berigan, 'Duke' Ellington and Fletcher Henderson, I believe we share the opinion of the majority when we say that for real enjoyment we do not have to stray far from home; and as for publicity, didn't we have Simone Simone?

A great deal of time and effort has been spent during this term in a Revision of the Constitution of the Medical Society. This action was precipitated by the desire of the local C.A.M.S.I. to become officially linked with the Medical Society. This required an alteration of the Constitution. As many Articles have outlived their usefulness, and many omissions have been noted, it was decided to make the first complete revision of the constitution since 1932.

A search through the records of the Medical Society Office revealed that nothing in the way of a constitution for the Medical Athletic Association was in existence. Each year over a thousand dollars is entrusted to this organization with nothing in writing to guide its expenditure. Hence the Constitution and By-Laws of the Medical Athletic Association has been drawn up by its executive, and has been approved by the Medical Society Executive.

In the Constitution of the Medical Society Article XVII is entitled *Medical Sick Benefit Fund*. Few students have even heard of this organization. From Section 2 of this Article, we quote: "The object of this Fund shall be to render financial assistance to subscribers who have incurred expenses owing to acute illnesses during the college term". This scheme sounds very attractive, but in the Constitution of 1932 no method of subscribing is suggested. From the Constitution of 1929 we find that subscriptions were obtained by selling "Medical Sick Benefit Fund Cards" to the students for $1.00. Later, this fee was added to the Medical Society Fee. More recently this $1.00 has been used as a subscription to the Medical Journal. Reading from the minutes of a meeting in October 1932, we find that as it was found inadvisable to raise the Medical Society Fee and the Medical Sick Benefit Fund was forgotten for the year. As far as we can tell from the minutes it has been forgotten ever since. Such a Student Health program as this, has been one of the basic principles behind the idea of C.A.M.S.I. Already it has given tuberculin tests free of charge to all Medical students. In our plans for the incorporation of this organization with the Medical Society, we feel that it will follow up the ideas that were behind the Medical Sick Benefit Fund and establish a Student Health Program on sound financial and healthful basis.

A report from a committee formed to investigate the possibility of holding an Annual Medical Society Banquet for all members of the Faculty of Medicine was approved by the Medical Society Executive. It was then suggested that this be included in the Revision to the Constitution for the approval of the students. In brief, the purpose of the banquet is to foster goodwill amongst the students. The time will be in the fall, to take the place of the Soph-Frosh banquet. The price, eased by a subsidy from the treasury of the Medical Society, would be fifty or seventy-five cents. The guest speaker is to be a prominent medical man and his subject to be a popular aspect of medicine. With support from all the students this function should become one of the real attractions of the year for every medical student.

To summarize the work done in the preparation of the Revised Constitution for the approval of the student body: four Articles have been added; forty-two minor changes have been made; one Article has been deleted; there has been a complete revision of the order of the Articles and an index of Articles has been added.

Preparations for *Daffydil* are now rapidly nearing completion as each year is engaged in the work for its skit. The show is a big undertaking and everyone engaged in its production deserves a great deal of credit. After the melodious preview we received at the Meds-At-Home, we can hardly wait to see the finished product finished.

SURGERY OF THE HAND

by

JOHN HAROLD COUCH, M.A., M.B., F.R.C.S. (Edin).

Department of Surgery, University of Toronto
and
Toronto General Hospital

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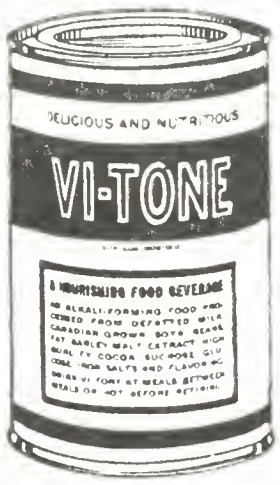

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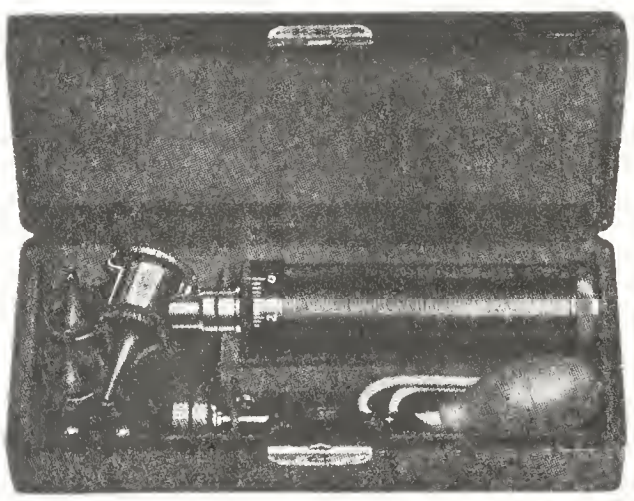

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